INDIAN SOCIETY FOR EMERGENCY MEDICINE

EMERGENCY MEDICINE

UPDATE

Proceedings of

THE SECOND NATIONAL
CONFERENCE ON EMERGENCY MEDICINE

19-21 MAY 2000

BANGALORE

EMCON 2000

EDITORS

DR. MABEL VASNAIK
DR. SHASHIRAJ E.
DR. BABU URUMESE PALATTY

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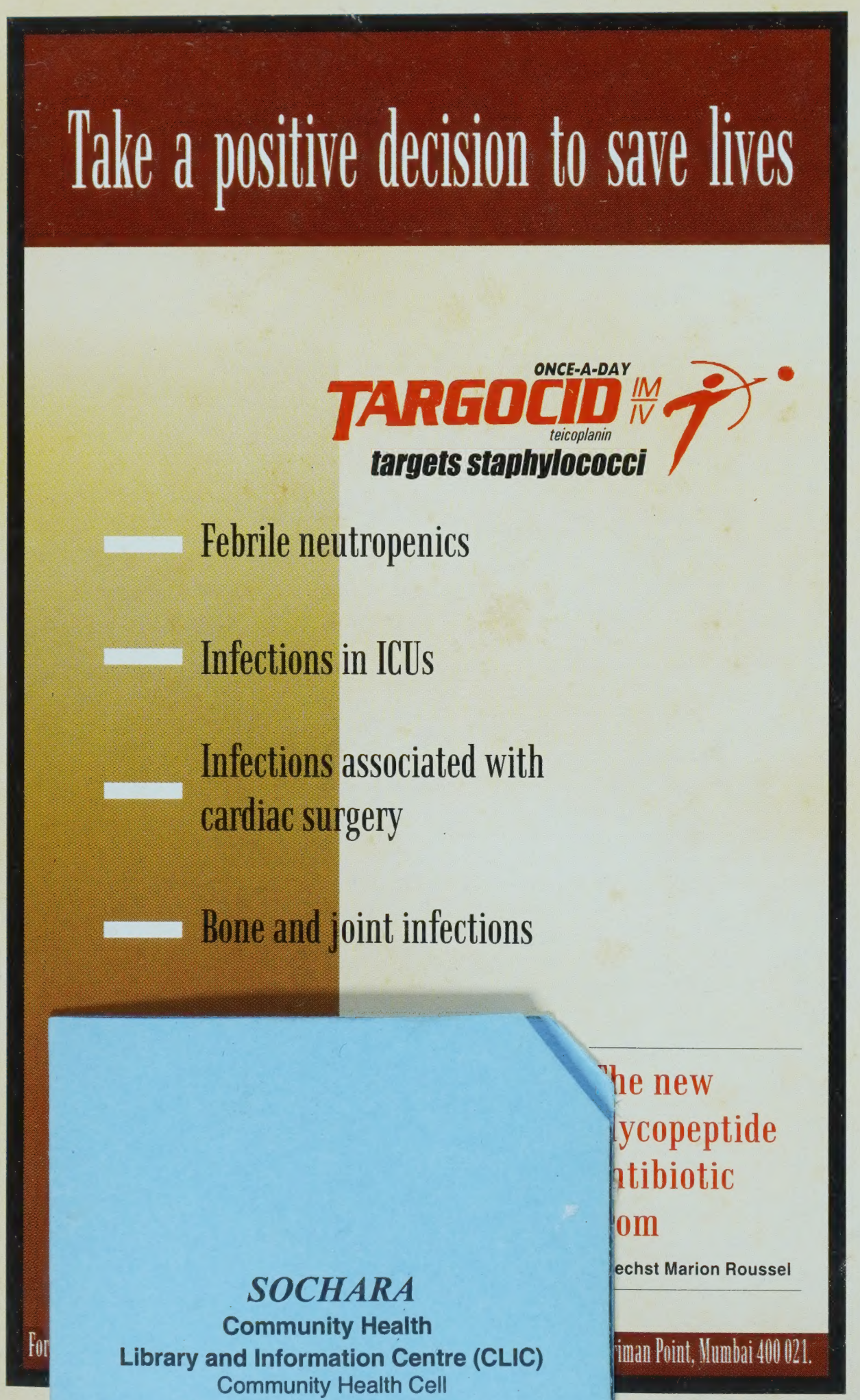

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Preface

It gives me immense pleasure to present this journal on the Proceedings of the Second National Conference on Emergency Medicine.

Emergency Medicine is a new, challenging and much needed speciality in this country. During this conference we hope to bring together doctors from all fields of medicine who are interested in developing this new speciality. We are overwhelmed by the response and support that was extended to us from all over the country while organising this conference.

The Scientific Committee has organised excellent scientific sessions where the management of various aspects of emergencies will be covered in detail by distinguished national and international faculty. This book contains the proceedings of all the scientific sessions of the conference and we hope it will benefit emergency physicians in their quest for excellence.

We are extremely grateful to the faculty who have contributed articles in order to enable us to bring out this journal.

We wish all of you a fruitful conference and a happy stay in Bangalore.

Dr. Mabel Vasnaik
Organising Secretary
EMCON 2000

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Challenges for Asian Emergency Medicine in the New Millennium: Aspirations and Expectations versus Availability

Dr V. Anantharaman

Department of Emergency Medicine, Singapore General Hospital, Singapore

A True Story

A sportswoman from the National women's hockey team sustained a head injury with a scalp laceration during an international hockey tournament in Singapore last month. A doctor at the site examined the sportswoman and requested that she be sent to hospital for immediate suturing of the laceration. The Secretary of the Hockey Federation rushed the lady in his own car through busy streets to a busy and well known Emergency Department. The summary of her management there was as follows:

	Arrival at Emergency Department
''''''''to''''''''''	Triaged by staff nurse. Had normal vital signs.
	Triaged as Priority 3 (Category 3- - ambulant, minor emergency).
''''''''to''''''''''	Attended to by doctor, had detailed clinical assessment, Skull x-ray done and films reviewed, after which toilet & suture of laceration ordered.
''''''''to''''''''''	Suturing done with aseptic technique in Minor Operating theatre.
''''''''to''''''''''	Patient left ED with medication and head injury advice.

The next day, the hospital received a stunningly critical letter from the Hockey Federation about the "long" waiting for the "emergency" patient they had rushed through the busy streets for immediate attention.

Though a simple episode, it illustrates the themes of this presentation very well, viz.:

Expectation: The Hockey Association Secretary expected immediate treatment and suturing on arrival for his national sports lady and was extremely upset that they had to wait.

Aspirations: The hospital was aspiring to provide good quality service and therefore

- triaged correctly
- assessed carefully and correctly and professionally
- x-rayed and reviewed the patient
- sutured the patient in an aseptic operating theatre. The procedure was done well.

The hospital was, however, unable to meet the operational service quality standards expected by the patient and the Hockey Association because:

Availability: On a busy afternoon with 8 resuscitation (Priority 1) and Critical care (Priority 2) patients and 15 Priority 3 patients arriving per hour, the bulk of medical and nursing staff had to care for the more acutely ill patients. Of course, the 2 Priority 4 patients arriving per hour had an even longer wait. Resources were limited and utilisation of resources had to be prioritised by medical rather than social needs.

Introduction

Emergency Medicine has often been presumed to be the prerogative of the developed world. All the

developments in this discipline are ascribed to work carried out in the United States, Canada, Australia and the United Kingdom. While this may be true in a vast variety of aspects of emergency care, the developing world and especially Asia, with its own cultural peculiarities and priorities, is reaching out to develop its own brand of emergency medicine. Obviously, since emergency medical care deals with humans, and humans are built somewhat similar the world over, the similarities would expectedly be numerous. In a rapidly shrinking world, there is a need for all of us to appreciate these similarities, yet accept that differences can occur in care practices, and allow for active exchanges of such cross cultural ideas. In this paper, I wish to provide an overview of the current status of Emergency medicine in Asia, especially in Singapore, and then go on to discuss the evolving status of this branch of Medicine and potential areas of emphasis for our continent.

Current Status

Asia, though having undergone a severe financial crisis, is still the fastest growing region in the world. The rapid changes in economic development had previously not been paralleled by appropriate improvements in the level of emergency care delivered to victims of illness and injury. In much of Asia, provision of emergency care has been characterised by the following:-

1) Lack of responsive and time-sensitive pre-hospital care systems: This has been owing to the frequent lack of a simple Early Access System in many countries of the region. The result has been a relative lack of use, by Emergency patients, of available Emergency Ambulances, which have over time been used more for inter-hospital transports and for non-emergency calls. Singapore instituted a single Emergency Ambulance and Rescue Access Number (995) more than 20 years ago and this has helped in the development of the country's Emergency Ambulance Services. Other countries have had a multitude of such numbers. However, over the last few years, more countries in the region are adopting single numbers. Country-wide standards of Paramedic Training care are currently being introduced in Thailand, Malaysia and Singapore. Local health and civil authorities are recognising the benefits that a dedicated Emergency Ambulance Service can bring to initial treatment of the acutely sick and injured. Medical direction is also being introduced, and has to be done with locally flavoured protocols.

2) Lack of categorisation of Hospitals: It has traditionally been a source of pride for many hospitals to accept the very sick and maintain the latest of equipment. Such impressive equipment have often not been paralleled by a proportional increase in the standard of medical care provided. The impact of absence of other support facilities and dedicated personnel has also often not been appreciated, even by health care workers and administrators. The financial crisis, of which we are seeing the tail end, has, hopefully made all of us realise the need to work together to optimise use of resources available in the community rather than expending limited funds in obtaining high cost facilities from expensive international sources. Hopefully, with proper guidance from an increasingly enlightened medical leadership and a health care executive that is feeling the financial squeeze, greater sense will prevail in the acquisition of expensive and sophisticated medical gadgets by hospitals. It is increasingly becoming obvious that categorisation of hospitals into equivalents of Levels 1, 2 and 3 centres and institution of community discipline in acquisitions and in arranging appropriate referral patterns by the pre-hospital care services will help to ensure better distribution and utilisation of scarce and expensive resources needed for initial patient care.

3) Minimal Care provided by many Emergency Departments: Unfortunately, there are many in this part of the world who still believe that patients will only get proper and definitive care in an in-hospital environment. Therefore, in many communities, Emergency Departments are only expected to perform the role of a traffic policeman, directing casualty traffic to the various inpatient departments. Many Emergency Departments are equipped with only the very basic equipment required to carry out simple patient examination and manage minor medical or surgical conditions. Inpatient care, though relatively expensive, is an easy option for the many who are not keen to make major decisions on initial management of patients. Sometimes easy availability of inpatient beds results in ambulant patients spending a number of days in hospital for the management of relatively minor wounds. However, more of the larger hospitals are increasingly being faced with the problem of inpatient beds not being readily available, and sick patients having to wait at busy Emergency Departments for hours on end. In Singapore, the gradual development of Observation Medicine over the years has resulted in the development of increasingly treatment oriented Emergency Departments. Similar developments have also ben noted in neighbouring Malaysia.

4) Emergency Medicine - a specialised medical discipline: Emergency Medicine was recognised as a distinct medical discipline by the Ministry of Health, Singapore in 1984. Since then, training posts have been created in the discipline. The first local three - year structured, basic post - graduate training programme in Emergency Medicine was started in 1989 to prepare candidates for the final examinations of the Royal College of Surgeons of Edinburgh in Accident & Emergency Medicine & Surgery. To date, 36 doctors from Singapore have successfully completed these examinations and returned to the country to assume senior positions in the various Emergency Departments. In 1990, an Advanced Training program was introduced to further enhance the level of skills of these trained Emergency Physicians in specific areas of care. The year 1996 saw the introduction of an intensive undergraduate training program in Emergency Medicine. Later this year conjoint examinations for the Fellowship of the Royal College of Surgeons of Edinburgh (Accident & Emergency Medicine & Surgery) and the Master of Medicine (Emergency Medicine), Singapore will commence in Singapore and this will open for Asian countries an avenue for achieving international acceptance and recognition for the doctors who will lead Emergency Departments in Asia well into the 21st century. A national training committee for Emergency Medicine oversees these academic developments in Singapore.

There have, however, been no similar parallel developments in the other countries of the region. Emergency medicine is not yet nationally accepted as a distinct Medical discipline in these countries. Resources are not allocated yet for the training of doctors, nurses and medical students in emergency care. There are currently no National Board Examinations in Emergency Medicine in any of the countries of South East Asia. The politics of recognition of training posts currently mean that for graduates of Malaysian and Singapore and, perhaps, Indian Universities, only the United Kingdom's Royal College of Surgeons of Edinburgh allows direct admission to the Emergency Medicine Board Examinations. Such direct entry is not available to similar examinations in Australia, Canada and the United States of America. Protectionism is still rampant, even in medicine, under the guise of protection of standards.

Goal of Emergency Medicine

The perceived goal of Emergency Medicine has been the early and rapid provisional diagnosis, in some

form, of medical and surgical emergencies (both trauma and non-trauma) so as to allow the early initiation of therapeutic manoeuvres or procedures in timely fashion. This is important to minimise morbidity and mortality and return the individual to a just and useful role in society cost-effectively.

More of emergency medical care is moving into the realm of high technology and costly medications and equipment. Lack of readily available finances prohibits the use of such high technology stuff often used in the developed world in the developing countries of Asia. The main problems appear to be:

1) lack of infrastructure for injury and disease control
2) poor enforcement of safety regulations
3) low level of access to emergency medical care
4) inadequate emphasis on training in core skills for front-line responders
5) a great belief in the essential need for hi-tech medicine as the panacea for the community's urgent health problems

The key towards achieving the goals of Emergency Medicine in Asia is to move towards a strong community foundation in basic emergency care with subsequent development of individual areas of Emergency care and emphasising cost effectiveness and efficiency.

Future Directions in Emergency Care for Asia

1) Academic Emergency Medicine: There is a need for Emergency Medicine to be granted status as a distinct medical speciality in the rest of Asia. With such recognition, programs can be developed to implement undergraduate and post graduate training in Emergency Medicine. Singapore has learnt many lessons in implementing such programs over the years. These lessons would be made available and can be used to develop individualised programs that are relevant to the realities of the terrain, geography and culture of our region. These could lead to the development of a post graduate diploma offered by the region for the regions nationals. Areas of commonality should then be established with similar programs in Australia, Canada, Great Britain and the USA, for example, with the aim of achieving cross recognition of each other's academic programs. In the rapidly shrinking world that we live in, this should remove many of the protectionist tendencies currently inherent in individual national emergency medicine training programs.

2) Emergency Nursing: One cannot expect to see continuous progress in the care of emergency patients if emphasis is only given to the training of doctors in Emergency Medicine. Our nursing colleagues, who do a lot of the leg work in the various Emergency Departments, are often neglected for purposes of training. They are equally important members of the Emergency Care Team. If we in the medical profession do not take up the case for nursing education and become advocates for good training programs in Emergency Nursing, it will become even more difficult to improve the level of emergency patient care in our communities. In Singapore, we began a one year Advanced Diploma Program in Emergency Nursing in 1995 in Singapore. After nearly five years and 75 trained emergency nurses we are beginning to see a keen interest to move the initial care of patients to greater heights. Some of our strongest advocates of good and rapid advanced care are our nurses. They are good allies to have in implementing and managing a number of the care programs that we may want to develop in our departments.

3) Advanced Post-Graduate Training: Emergency doctors in much of the region are often not regarded as experts in the various areas of emergency care. Therefore, a cardiologist is often regarded as the expert

in emergency cardiac care, including pre-hospital cardiac care, even though the guy who works closely with the ambulances or in training ambulance crew may happen to be the emergency physician. A surgeon is often consulted about the initial basic care to be given to trauma victims in the field, even though he may work less often than the emergency room physician in the pre-hospital environment. Too often, even emergency physicians do not have the strength of conviction in the areas they are supposed to be the most familiar. We attempted to address these issues when we introduced Advanced Emergency Medicine Training nearly 10 years ago. The objective of advanced training was to allow the recently board-certified emergency physician to gradually nurture into an independent, confident senior physician with true in-depth knowledge of specified areas of interest in emergency medicine. This person would still be a good general emergency physician. However, he would be extremely knowledgeable and skilled in one or more of the following:

a) Emergency Cardiac Care
b) Emergency Trauma Care
c) Emergency Toxicology
d) Emergency Paediatrics
e) Emergency Pre-hospital Care
f) Disaster Medicine
g) Emergency Observation Medicine

The fellowship programs we are developing in these areas of interest would form the basis for these advanced training systems. In addition to the training that could be provided in Singapore, we attempt to send these senior doctors to centres in other countries that are actively involved in these identified areas of care. This allows the advanced trainee a broader perspective of the specific interest area. Such an educational experience can only contribute towards the development of Emergency Medicine experts in these areas of interest. This will also assist in improving the standards of care for patients with problems relevant to these areas of interest. This is also a precursor to the development of subspecialities within the main discipline of Emergency Medicine. At the Singapore General Hospital's Department of Emergency Medicine, we have established to-date five sub-speciality branches, viz. in Emergency Cardiac Care, Emergency Trauma Care, Emergency Toxicology, Disaster Medicine and Pre-hospital Emergency Care. These branches are developing their own programs. To allow greater scope for development and success, senior doctors and nurses with interest in these areas have been grouped and tasked to achieve targets set by themselves. These programs could become future avenues for the training of the regions Emergency Physicians in the specified areas. They could also be vehicles for International Exchange Programs in Emergency Medicine.

4) Pre-hospital Emergency Care: The next decade should be spent in building up the foundations for a strong basic level of pre-hospital emergency care. Such a basic foundation will be based on the following principles:-

a) obtaining a commitment from local health authorities that they are prepared to work together and work towards a unified pre-hospital care service.
b) identifying the main provider of pre-hospital emergency care.
c) developing a system of easy access to the Emergency Medical Services
d) establishing a system for medical despatch, standards of despatch instructions and identifying requirements for training of despatchers.
e) developing an integrated communications system with ambulances, ambulance stations and receiving hospitals.
f) incorporating a system of medical direction into the operations of the Emergency Ambulance Service.

g) drawing up and implementing systems of identifying various categories of sick or injured persons.

h) developing transport protocols and criteria to send patients appropriately to different levels of hospitals.

i) implementing a quality management system.

j) working towards a system of maintenance of ambulances and ambulance equipment.

The achievement of the above will be attempted through an upgrading of available resources and networking of available agencies and personnel. An investment in training will have to be made. Such an investment will go towards developing a training cadre and training system. The training content should suit the local environment and local language. In addition, there will be a need to build a good foundation of public consciousness of life-saving first aid, including CPR.

For Singapore, developments in pre-hospital care over the next decade will include, in addition to the above:

a) A system of electronic data communication to facilitate transfer of information from ambulances to receiving hospitals. Called the HEAL (Hospital & Emergency Ambulance Link) System, it will aim to simplify the generation of an Ambulance Case Record and alert hospitals early of every arriving ambulance. A pilot run of this system was begun in late 1998, and has been successful. We are now working towards nation-wide implementation of this system.

b) Emphasis on special areas of care that are time sensitive, such as chest pain and myocardial infarctions and stroke.

c) Automating the audit of all ambulance runs.

5) Observation & Fast Track Medicine: The pressures of increasing health care costs, non-availability of inpatient hospital beds and increasing use of Diagnosis Related Groups (DRG) to cut governmental health expenditure are forcing Emergency Departments to consider more cost efficient means of managing a wider variety of emergency patients, such as on an ambulatory basis. In doing so, however, a greater degree of standardisation is being demanded in management of many emergency conditions. This may, inadvertently, be a boon for the greater development of a more aggressive form of emergency care. Heart ERs are an example of this. We have initiated moves to develop protocols for 6 to 8 hour short stay chest pain units, initial management of congestive heart failures, emergency relief of a variety of arrhythmias using inexpensive treatment regimens, severe hypertensives and pulmonary embolism patients. With a more rapid and aggressive diagnostic work-up of such patients and very early initiation of definitive therapies, there is expectation of lesser inpatient admissions and a shorter duration of morbidity for those who are hospitalised. Many patients with minor head trauma, a variety of acute abdominal complaints and acute bronchial asthmatics are potential beneficiaries of such fast-track systems. Such systems will entrench the very important role of Emergency Medicine in the care of the acutely sick and injured.

6) Development of Triage Systems: Triage in Emergency Departments has often been done in an extremely cursory manner. This has led frequently to a less-than-optimal sorting of patients presenting at the shopwindow of hospitals. We need to develop practical - oriented Triage training program for budding Triage officers. These programs widen the early use of rapid diagnostic resources to increase the accuracy of triage, and reinforce the belief that triage should be done not by the most Senior Surgeon available, but by a trained triage officer. The senior surgeon could perhaps contribute more by using his practical procedural skills in the treatment of patients. Greater accuracy in triage

would be likely to result in more accurate definition of the area or track that a patient should follow in the Emergency Department. Accurate triage would also be the start point for the patient to be entered into the various clinical pathways that may have been developed in the Emergency Department. We need to pay greater attention to increasing the efficiency of triage in our departments. It holds the key to speedier and more cost efficient management of the variety of patients that we encounter daily.

7) Care Process Re-engineering: An Emergency Department can be regarded as a mixture or combination of a variety of care processes. Many of the operational and clinical processes that have come about over the years have been owing to the personal preferences of various staff. We often do not question various such practices. However, the various social pressures that are currently pushing the pace of change in Emergency Departments also demand a review of the way we have been managing the various processes in Emergency Care. Change is a difficult thing. It is, however, necessary to keep up with the persistent increasing expectations on the part of patients and of changes in medical practice. Change, if implemented in a top - down fashion often meets with resistance from various grades of staff. Change is often easier to implement if staff feel ownership of the changed process. We find that the principles of Business Process Re-engineering can be applied in the Emergency Department setting to improve the quality of the care that we provide.

The objective of process re-engineering in Emergency Departments is to improve the quality of the care provided by standardising and improving the level of service, minimising administrative activities of patients and staff and minimising movement of patients, relatives and staff. Getting departmental staff involved in the care process re-engineering effort is one way of instilling a culture of continuous quality improvement within the Emergency Department. It may help to cut patient waiting and process times.

8) Performance Standards and Quality Management: Quality management in Emergency Departments has often been a sore point with many members of the staff. The lack of internationally accepted and clear markers of performance of EDs has compromised the ability of some departments to obtain the resources required to provide what they consider to be an optimal level of service. It is important that we consider agreeing on common indices of performance. Performance measurement is however, fraught with difficulties. I would like to suggest that Emergency Departments consider five different performance indices, viz.

a) Operational Index - refers to the ability of a department to provide a seamless service. A reasonable measure of operational performance in an Emergency Department would be the Patient Turnaround Rate (PTR) reflected as the average number of patients seen over a one hour period.

b) Service Index - This refers to the provision of a personalised service by the department to the patient. Derivation of a Patient Satisfaction Rating (PSR) needs to be considered.

c) Organisational Development - A well developed and managed organisation refers to one that has attained a level of maturity to the effect that the staff enjoy providing the service for which they have been employed. A Staff Satisfaction Rating (SSR) may reflect the status of organisational maturity, management and development.

d) Clinical Quality - is an extremely difficult entity to measure. Often, mortality statistics are used. However, mortality is only the tip of the iceberg. Outcome studies are assuming greater importance in measurements of clinical quality. If the clinical

function of an Emergency Department can be defined by three roles, viz. the ability to discharge patients accurately (which may perhaps be reflected by the reattendance rate for the same complaint within three days), the ability of the department to admit accurately (which may perhaps be denoted by early discharge rates of ED admitted inpatients (EDR), and the ability of the department to manage patients well during their stay in the ED. Though there is no golden index available for measuring this, aggressive and rapid diagnosis and initiation of early treatment can potentially result in better outcomes for the patient. One may wish to monitor the Average Length of Stay (ALOS) as an indicator of quality of emergency treatment. These three rates may be used by EDs to derive a Clinical Quality Index.

With the above indices monitored on a regular basis it would be possible to have an overall indicator of performance quality, say the Department's Quality Index.

e) With all of the above it may be theoretically possible to derive indicators of Financial Performance. The financial performance of a department may be reflected as either the `Cost Benefit Index', which should be the mean cost per patient seen per unit. Departmental Quality or the Productivity Index of an Emergency Department may be reflected as the Revenue earned per ED staff. With such indices, hospital administrators would have a truer picture of an Emergency Department's performance when considering resource allocation for an improved level of performance.

9) Care Plans and Clinical Pathways: Various factors enumerated earlier are pushing for a more efficient and low cost system of emergency medical care. It can be reasonably presumed that costs are higher and inefficiencies greater if we allow for all the individual variations in styles, habits and preferences in institutional Emergency Medicine practice. In addition, individuals differ in their attention to detail, and degree of personalised care that they bring to their patients. All these will reflect on the standards of medical care in the organisation. Certain minimally acceptable standards of care have to be established. There could be minimal standards for assessment, diagnostic procedures, treatments and quality of care. These can be formalised by the development of written care plans and clinical pathways for use by both medical and nursing staff in Emergency Departments. Such development requires a lot of time and effort, but once complete they provide a template for standardising management of various clinical problems, especially when such management provides for safety in clinical practice. It also provides a mechanism for audit of the clinical care provided, and a useful teaching tool in the training of Emergency Medicine residents and undergraduates.

10) National Societies for Emergency Medicine: Over the last ten years, a number of National Societies for Emergency Medicine have been set up in Asia. The Indian Society for Emergency Medicine is one such organisation and together with sister societies in Asia will be working hard to work out common solutions to common problems and to help each other in working out low-cost and efficient solutions to the various problems affecting the provision of Emergency medical care to the sick and injured in Asia. The Societies must also seek to work together with sister organisations in the other parts of the world and foster closer co-operation so as to achieve commonality in standards of medical practice. Areas where a close working relationship may be established could be in post-graduate exchange programmes, joint examinations, joint scientific meetings, joint research projects and joint development of clinical protocols for a variety of common clinical problems.

Resources

To achieve the various things that we have set out to do, what resources are we going to draw on? We need to make a few commitments:

a) **People.** We need to train and nurture our doctors, nurses and other emergency care givers. Therefore, we need to make commitments in training. Training is time consuming. Training is the basis for long-term change and should be one area for commitments. Private industry that draws upon the resources of a large part of the work force must be seen to have a major contribution to the training of health care staff who will provide emergency care to the workforce and who will try their best to return the sick and the injured to a productive role in society as quickly as possible. Therefore set up the system of training and recognition and get private industry to become a vital force in the training mechanism.

b) **Funds.** These are always in short supply. Money needs to be used wisely. If we allow funds to be siphoned off for personal gain, our society will be the loser. Eventually, that loss will also affect us adversely. Whether we like it or not, a little bit of altruism is important in the carrying out of our daily activities. We need to be constantly aware of the greater good that can be achieved if every dollar or Rupee can be stretched to achieve maximum results. If we can accept that the responsibility for emergency health care is to be shared by three groups, viz. the individual, the state and the employer, then mechanisms can be worked out to ensure that emergency care can be made accessible to all in the community. Low-cost community health insurance schemes underwritten by the state and private industry should be looked into to increase the reach of affordability of emergency care.

c) Medical organisation, equipment and supplies. I have stated earlier that expensive equipment purchased from other countries does not equal superior medical care. We must allow and facilitate the development of local home based industry to cater to providing the bulk of our medical equipment and supplies. Of course, we must all be ever cognisant of product quality. Home based industry would allow low cost supplies. It will also increase the scope of employment for our people and will be a gain for the economy. It is obvious that positive economic development is a vital factor in improving the state of health of a community and the standard of medical care.

Conclusion

As we are into the new millennium, we will need to take charge of the development of our Emergency medical systems. While we will adopt an "Asian solutions for Asian systems" approach, we are all too aware that in this rapidly shrinking world, interdependence, sharing of ideas and solutions and a willingness to not only work with one another, but also to help train each other, regardless of continental boundaries, is the way forward.

We must continue to dream and to aspire to provide a higher level of emergency care to our community citizens. Our communities' expectations of the level of care that we should continue to provide will increase and perhaps at a greater rate than we can cope with. The long term solution lies in training and organisation. We must also always be aware of the limitations that exist in terms of funding and other resources. Therefore, we need to move towards maximal utilisation of scarce resources and towards working out low cost solutions to our various emergency health care problems. Look carefully at what you have in your own community and you will discover the invaluable resources that you should be able to utilise for the common good.

Acute Respiratory Distress Syndrome

Barry A. Harrison, M.D.
Mayo Clinic and Foundation, Rochester, MN 55905, USA

Objective

To better understand the acute respiratory distress syndrome (ARDS) as a syndrome precipitated by many diverse factors with the aim that prompt recognition and treatment aims to prevent ARDS progression. To understand the pathophysiology that has lead to the current ventilator support strategies, that have decreased the mortality in ARDS.

Introduction

During the Vietnam War era, administration of blood and crystalloid/colloid was widely implemented, not only in the military setting, but also in the civilian setting. With this new treatment, however, came the recognition that patients were now developing severe respiratory failure secondary to extravasation of fluid from the pulmonary-capillary bed into the lung interstitium and into the alveoli. These patients had a condition that was different from the respiratory distress due to pulmonary edema that patients with heart failure developed; these patients did not have elevated pulmonary capillary wedge pressure. Patients had hypoxemia and diffuse infiltrates on chest x-ray, but the use of 100% oxygen did not correct the hypoxemia because the cardiac shunt through inadequately ventilated parts of the lung was too large. This "new" condition was termed initially the "acute respiratory distress syndrome" by Ashbaugh, et al. in 1967 (2). Subsequent descriptions of the syndrome used "Adult Respiratory Distress Syndrome" to distinguish it from neonatal respiratory distress syndrome which is a condition due to a surfactant deficiency.

Definition

Over the last 25 years, it is recognized that adult respiratory distress syndrome or acute respiratory distress syndrome (as it is now known) is caused by a number of factors. The most common etiologies, however, are trauma, infection, shock, sepsis, and pulmonary aspiration of gastric contents (3,4). The recommended criteria for the diagnosis of acute respiratory distress syndrome is a recognized precipitating etiology that is acute in onset, a PaO_2/FiO_2 ratio less than 200 mmHg, bilateral infiltrates seen on frontal chest radiograph and a pulmonary artery wedge pressure less than 18 mmHg, or no clinical evidence of left atrial hypertension.

Epidemiology

The incidence of ARDS is approximately 9.6 per 100,000. The incidence will vary depending upon the presence and type of trauma and illness within the study population. Once acute lung injury develops, patients' survival depends on the severity and extent of the lung injury and, to a certain extent, how meticulous we as health-care providers are in managing these patients. Up until the early 1990s the mortality rate from ARDS was between 50 to 60%. At present there are case series from single institutions and randomized prospective trials with reported mortality of 30%. Early in the syndrome patients will die from hypoxemia, but more commonly during the later stages the majority will die from the associated organ dysfunction and failure. Importantly for survivors lung function returns to almost normal within one year.

Pathogenesis

ARDS is not necessarily the result of discrete injury to the lung. More commonly, the lung is one of several organs that are injured. This injury is the result of an initial event (infection/trauma) that activates a number

of host defense mechanisms. The excess activation of these defense mechanisms leads to a situation in which the patients' own tissues are injured. This diffuse, and self-directed, systemic inflammatory response syndrome (SIRS) can lead to dysfunction in several organs, leading to the development of multiple organ dysfunction syndrome (MODS) (5). The lung, for a variety of reasons, is one of the most frequently affected organs, one that is injured early in the development of MODS. The systemic mediators of these syndromes (ARDS, SIRS, MODS) are products of neutrophils, complement, and other blood-borne elements such as platelets.

The scenario for neutrophil-induced ARDS includes activation of neutrophils by an infectious agent, aspiration of gastric content, or other process. The activated neutrophils sequester in the lung (and other organs) and release superoxide anions that destroy or injure the pulmonary endothelium. With the injury to the pulmonary epithelium and endothelium (the semipermeable barrier that exists throughout the lung), fluids and occasionally protein extravasate into the interstitial space and alveolus. The same scenario can be constructed for complement and platelets. There is ample evidence in the literature, however, that the actual event is multifactorial and all these elements (neutrophils, complement, and platelets) are involved in the development of acute lung injury (6-8).

Pathophysiology

In ARDS pulmonary edema results from the abnormal accumulation of extravascular water in the lung. Hypoxemia results from perfusion of atelectatic and fluid filled alveoli. The right to left shunting of blood flow may approach 25-30% of the cardiac output (9). High airway pressures result from mechanical ventilation due to decreased chest-lung wall compliance (<20 ml/cm H_2O) and from an increase in airway resistance. Decreased lung compliance is really a reflection of pulmonary edema and atelectasis. With the increase in dead space ventilation the net effect of these abnormalities is to increase the work of breathing. In ARDS the work of breathing may be responsible for 25-50% of the body's total oxygen consumption. Pulmonary artery pressure is increased due to alveolar hypoxia and the presence of vasoactive mediators such as thromboxane. Intravascular obstruction from platelet thrombi and or perivascular edema may also contribute to pulmonary artery hypertension.

Management

The management of ARDS is outlined in Table 1.

Table 1. Management Therapies for ARDS

*	Prevention
	Excess intravascular volume
	Gastric aspiration
	Adequate resuscitation of shock state
*	Identify and Treat Precipitating Event
*	Ventilatory Support
*	Analgesia/sedation/neuromuscular blockers
*	Hemodynamic support
*	Experimental Therapies

Ventilatory Support

The FiO_2 should be set no higher than 0.6 unless higher levels are required to maintain the SaO_2 greater or equal to 88%. PEEP has been used in the management of patients with ARDS for the last two decades (14). PEEP maintains the functional residual capacity (FRC) by keeping alveoli open, preventing their collapse at end expiration. The continuing collapse and expansion of alveoli with each VT, as can occur when PEEP = 0 cm water, has also been thought to lead to volutrauma.

Current treatment with PEEP would indicate that peak levels be maintained between 10 and 15 cm of

water pressure, whatever is adequate to maintain or improve arterial oxygenation (SaO_2>88% on FiO_2<0.6), without compromising venous return to the heart decreasing cardiac output.

The role of mechanical ventilation in these patients has also evolved considerably over the last 25 years. Initially, mechanical ventilators were "pressure triggered." Once the defined pressure was achieved then the positive pressure would trigger "off." This was problematic initially when managing patients with ARDS because as the patients' lung compliance worsened, i.e.., the lung got stiffer, VT decreased and the patient was under-ventilated. This served as a stimulus for the development of volume-cycled ventilators so that a known VT would be delivered to the patient, independent of the airway pressure necessary to deliver the VT. We now know that these increased airway pressures and VT may actually contribute to damaging the lung. Because parts of the lung may be normal (as already commented upon) and other parts may be very edematous and stiff, the large VT goes into the relatively normal (compliant) part of the lung over-distending and damaging that portion (15). New approaches to mechanical ventilation, therefore, are targeted at decreasing the VT and maintaining adequate oxygenation. The pendulum has swung back to the point where we now use pressure-triggered ventilation (pressure controlled ventilation). By using this technique, we can be sure that the pressure will not be excessive. In the situation where there is under-ventilation and the patient develops retention of carbon dioxide, there is often no reason to change the patient's therapy. In fact, this hypercapnia may actually be desirable and some clinicians actually advocate "permissive hypercapnia" (16,17) in the management of patients with ARDS, with the sole goal of maintaining low inspiratory pressures and low VT. As long as the pH is not excessively low (less than 7.20), there is no convincing evidence that this therapy is wrong. It may, indeed, be beneficial, though this has not yet been proven by randomized studies.

A recent trial by the ARDS network, sponsored by the NIH demonstrated decreased mortality, absolute level 30% and increased mechanical ventilator free days in patients randomized to receive 6 ml/kg tidal volume versus 12 ml/kg tidal volume.

Analgesia, Sedation and Neuromuscular Blockers

Titrated amounts of analgesia and sedation is necessary in the treatment of ARDS to provide anxiolysis, amnesia, depress the increased ventilatory drive that leads to increase work of breathing and also to allow the patients to tolerate the modes of mechanical ventilation and PEEP. The Society of Critical Care Medicine has issued guidelines for analgesia and sedation in the intensive care. They include the use of fentanyl and morphine as analgesic agents and lorazepam as a sedative. Their use in combination is synergistic both for the clinical effects and also their side effects. The use of neuromuscular blockers in ARDS patients has anecdotally said to be decreased, the main reason cited is prolonged neuromuscular blockade, and myopathy. However, their use is still indicated, particularly with the use of inverse-ratio ventilation, high PEEP, permissive hypercapnia, and other experimental strategies. Critical care illness polyneuropathy is another neuromuscular disease that may occur in ARDS patients, but this is independent of the use of neuromuscular blockers and related to SIRS and MODS affecting the peripheral nervous system.

Hemodynamic Support

In animal models, fluid accumulation in lungs with increased vascular permeability occurs at lower pulmonary capillary pressures than in normal lungs. If lung microvascular pressure is elevated in the presence of an increase in lung vascular permeability, then the quantity of pulmonary edema increases substantially.

Animal models demonstrate that the addition of PEEP or the administration of albumin does not decrease extravascular lung water. However, phlebotomy, nitroprusside and furosemide are associated with a decrease in extravascular lung water.

Clinically, retrospective studies have reported increased survival in ARDS patients who had in one study a negative fluid balance or in another a 25% reduction in pulmonary artery wedge pressure. A prospective study utilizing extravascular lung water measurement and pulmonary artery catheter measurements to control extravascular lung water as the treatment arm demonstrated a significant decrease in extravascular lung water, shorter duration of mechanical ventilation and stay in the ICU. However, the survival rate was the same in both groups and the study population included permeable and non-permeable pulmonary edema.

Thus, although excessive fluid administration is probably detrimental in ARDS, sufficient fluid administration is important to maintain tissue perfusion and oxygen delivery. The empiric use of diuretics has not been proven. With the "Last Hurrah" for the pulmonary artery catheter (PAC) announced at this meeting the following are suggestions for the use of the PAC in ARDS patients.

- The use of the PAC is not mandatory in the management of ARDS patients
- To confirm diagnosis, especially in patients with coexisting cardiac disease
- Where there is difficulty in managing fluid balance, particularly if sepsis or renal failure is present
- Titrating inotropic, vasopressor, vasodilator and PEEP therapies

Experimental Therapies and Strategies

Table 2 outlines experimental strategies and therapies that are now seen in the support of ARDS.

Table 2. Experimental Strategies and Therapies for ARDS

A. Increase Oxygenation
   * Reverse I:E ratio
   * Prone position
   * Inhaled nitric oxide therapy
   * Partial liquid ventilation with Perflubron
   * Extrapulmonary gas exchange
      a. Extra-corporeal - ECMO, ECCO2R
      b. Intra-corporeal - IVOX

B. Decrease Volutrauma
   * Permissive hypercapnia
   * High frequency ventilation
   * Tracheal gas insufflation
   * Computerized controlled ventilator management

C. Decrease inflammation
   * Corticosteroids
   * Surfactant
   * Omega-3 fatty acids

A. Increase Oxygenation

Inverse ratio ventilation

New modes of mechanical ventilation also include inverse ratio ventilation (IRV) (18). Normal inspiration is relatively short compared to expiration with a ratio of approximately 1 : 2 or 1 : 3. In a very stiff lung, trying to deliver even a small, inspired VT, however, may require excessive inspiratory pressure. By delivering this VT over long periods, alveoli may be recruited with lower peak inspiratory pressures. This is achieved by reversing the ratio of inspiration to expiration from 1 : 2, for example, to perhaps 1 : 1 or even 1.5 : 1.0 or higher. IRV is another indication for using neuromuscular blockers in managing these

patients because patients will not tolerate this particular mode of mechanical ventilation unless they are either very comatose or unless we administer a neuromuscular blocker.

Prone position

Patient position may also be an important patient care factor. Though in the past the pulmonary injury was thought to be distributed evenly throughout the lung, studies of computed tomographic scans have demonstrated that frequently the injury is not homogenous; there are areas of the lung that are worse, some that are better (10,11). You may find that during positioning of a patient, oxygenation may worsen. If that is the case, return to the previous position might be beneficial. Conversely, if the patient worsens, a change in position may help (12,13).

Inhaled nitric oxide

Nitric oxide (NO) is a selective pulmonary vasodilator (25). In 10 patients with severe ARDS, Rossaint et al. administered inhaled NO in concentrations of approximately 18 ppm, and achieved statistically significant decreases in pulmonary artery pressures and intrapulmonary shunt, while the PaO_2/FiO_2 ratio increased without changing the mean arterial pressure and cardiac output (26).

Liquid perflubron

A new mode of mechanical ventilation developed by Alliance Pharmaceuticals in San Diego, California, is Liquivent, i.e., liquid perflubron. This is an inert chemical compound in which oxygen is highly soluble. In the current clinical trial, perfluorocarbons are instilled into the lung to fill functional residual capacity, and the patient is ventilated with supplemental oxygen (23). The perfluorocarbons, by allowing oxygen to diffuse across fluid-filled alveoli and by improving lung compliance (because of a surfactant-like property), decrease airway pressure and improve oxygenation. Though the preliminary results are encouraging, the phase II, randomized, controlled trial has just begun.

Extrapulmonary gas exchange: extracorporeal

Extracorporeal membrane oxygenation (ECMO) was tried in a multicenter study in the 1970s, but it was not found to be beneficial (19). New developments in the utilization of ECMO have encouraged investigators at the University of Michigan to recommend ECMO for discrete subgroups of patients with ARDS. Gattinoni, in Milan, Italy, decided that the difficulties with ECMO were secondary to the anticoagulation that was required, and any benefits from ECMO were outweighed by the risks of coagulopathy and increased bleeding. He, therefore, developed extracorporeal carbon dioxide removal (ECCO2R) in which blood from the body has carbon dioxide removed extracorporeally (20). There is some oxygenation that occurs, but it is not total cardiopulmonary bypass. Though his results were encouraging, a more recent study by Morris, et al. in Salt Lake City, Utah has failed to demonstrate an improved outcome (21).

Extrapulmonary gas exchange: intracorporeal

J. D. Mortensen, an emeritus surgeon at the University of Utah in Salt Lake City (22), first developed the intravenous membrane oxygenator (IVOX). Though the initial results were encouraging, no controlled, prospective, randomized study demonstrated benefit from the IVOX. Although the oxygenator did what it was designed to do, if the underlying lung injury was too severe, it was irreversible and the patient eventually succumbed from complications of the underlying illness even with support of systemic oxygenation.

B. Decrease Volutrauma: High frequency ventilation and permissive hypercapnia

Newer therapies under investigation include the use of permissive hypercapnia (which was described previously) and high frequency ventilation. Both of these

therapies have as their main goal the limitation of airway pressure and volutrauma, with maintenance of oxygenation and higher carbon dioxide levels than normally would have been tolerated in the past. No benefit has yet been demonstrated for these therapies.

C. Decrease Inflammation

Oxepa - omega 3 fatty acids

There is also evidence that - because endogenous mediators are so important in the development of ARDS - manipulation of these same endogenous mediators by nutritional formulas containing high amounts of omega-3 fatty acids and anti-oxidants will improve oxygenation, and decrease airway pressure, and, at least in animals, improved outcome (24).

Surfactant

Unlike neonatal respiratory distress syndrome, surfactant is present in normal amounts in ARDS, though it may be dysfunctional. Clinical trials of exogenously administered surfactant in ARDS have not been encouraging (27).

Late stage corticosteroids

Large multi-centred trials comparing high dose corticosteroids in the 1980's in the treatment of ARDS found no benefit. Recently small trials have compared high dose corticosteroids at the ten to fourteen days following ARDS onset and reported increased survivorship. However the investigators carefully screened out any patient with infection, the trial was a cross over in design, and the numbers extremely small.

Conclusion

ARDS is a systemic disease manifested by non-cardiogenic pulmonary edema of multifactorial etiology. Prevention is important because, once ARDS develops, mortality is high. Treatment includes controlled FiO_2, PEEP, and ventilator techniques to decrease airway pressures and VT. Several new therapies are under investigation to try to decrease the high mortality rate associated with this disease.

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Acute Pulmonary Embolism

Gerard S. Kamath, M.D.
Mayo Clinic and Foundation, Rochester, MN 55905, USA

Perioperative Pulmonary Embolism

Surgery may result in an embolization of venous thrombus, fat, air, gas, tissue, or foreign substances. The occurrence ratio of some types of perioperative embolization have been determined by the use of transesophageal echocardiography (TEE), but, it is unclear how many of these episodes translate into clinically significant events. The causes, clinical presentation, pathogenesis, and management depend on:

A. The site of embolization

B. The rate of embolization

C. The nature of the embolized material

D. The extent of pulmonary vasculature involved

E. Previous cardiovascular status of the patient

F. Activity of reflex and humoral factors

The Pulmonary Considerations

1. The normal pulmonary vascular reserve capacity is such that about 50% of the cross sectional area must be lost before sustained pulmonary hypertension is seen. The normal mean pulmonary arterial pressure is about one sixth to one eighth of the mean systemic pressure and the pulmonary vascular resistance is about 10% of the systemic vascular resistance. Acute increases in right ventricular afterload as seen in massive pulmonary embolization lead to acute right heart failure. The maximum pressure that can be sustained by the right ventricle is 40 mmHg and beyond this the ventricle dilates and fails. The consequences of acute dilatation of the right ventricle are:

   A. Shift of the intraventricular system to the left limiting left ventricular filling.

   B. Further limitation on left ventricular filling by the restraining action of the pericardium.

   C. Tricuspid and pulmonic valve regurgitation

Effects of Pulmonary Embolism on the Lung

Abnormal pulmonary gas exchange occurs in a majority of cases of acute pulmonary embolism. This is evidenced by:

A. A widened AaO_2 gradient

B. Hypocapnia

The abnormal gas exchange in patients results from a variety of different mechanisms that include:

A. V/Q mismatches

B. Diffusion impairment

C. Mixed venous hypoxemia

D. Intracardiac shunting

When the embolitical material is air, fat, or amniotic fluid, vascular damage is significant with subsequent changes in permeability of the pulmonary vasculature leading to pulmonary edema at normal hydrostatic pressures (ARDS).

Changes are also seen in lung mechanics. The most common abnormalities are:

A. Increased airway resistance

B. Fall in static and dynamic compliance.

Paradoxical Embolization

Cohnheim first described systemic embolization via an intracardiac right to left communication in 1877. For this to occur, four conditions must be met:

A. Thrombus must be present in the venous system.

B. An abnormal communication must exist between right and left circulation

C. Clinical, angiographic, or pathological evidence of systemic embolism must be present.

D. A pressure gradient that promotes right to left shunting must exist at some time in the cardiac cycle.

A review by Lascalz of 25 cases of paradoxical embolism indicated that 18 were associated with PFO, 3 with ASD, 3 with pulmonary arteriovenous fistulae and one with VSD.

Deep Vein Thrombosis

It is estimated that DVT occurs in up to 5 million patients each year in the United States. Of these, 10% will have a pulmonary embolus and 10% of these will die. Of those who succumb to an acute pulmonary embolism, the majority will die in the first hour after the event. Without adequate treatment, those who survive are liable to have a recurrence and die as a result. It must be emphasized the pulmonary embolism is a complication of DVT and unless the emphasis is on preventing DVT, recognizing it early, and treating it appropriately, we will make little headway in impacting on the high mortality associated with pulmonary embolism. Ninety percent of all chemically significant pulmonary embolisms arise from thrombi proximal to the popliteal vein. However, most of these represent propagation from calf vein thrombi. Thrombi arise de novo in the proximal veins due to direct intimal damage near the saphenofemoral junction as seen in hip fractures and hip surgery or surgery near the groin area. Central vein catheters are an increasing source of thrombi that embolize.

Diagnosis of DVT

Duplex scanning has rapidly become the noninvasive procedure of choice for diagnosing DVT. Other methods include:

A. Contrast venography

B. I^{125} fibrinogen uptake test

C. Impedence plethysmography

Incidence of DVT in Surgical Patients

Venous thromboembolism is rare during the preoperative and in the operative period. In the postoperative period, the incidence is particularly high for orthopedic and neurosurgery, major trauma and pelvic surgery. The major risk factors include age greater than 40, previous thrombo-embolic event, preoperative hospitalization greater than 6 days, immobilization after surgery, perioperative transfusion greater than 1 unit, presence of leg ulcer, and previous major orthopedic event. Additionally, previous medical problems like malignancy, recent MI, congestive heart failure or renal failure increase the risk of DVT. Proteins C, S, and antithrombin III deficiencies must be looked for in the younger patient with DVT or if there is a family history of DVT.

Diagnosis of PE

Pulmonary angiography is the gold standard for diagnosing PTE. Spiral CT scanning is showing increasing promise. The most common test performed is the V/Q scan. The sensitivity and specificity of this mode of diagnosis has been determined in the PIOPED study. The accuracy of V/Q scans may be improved by combining it with significant clinical indicators.

Intraoperative Presentation of PTE

Pulmonary embolism is difficult to distinguish from the cases of acute hemodynamic deterioration. Typical signs and symptoms in anaesthetized patients include hypoxemia, cyanosis, hypotension, wheezing, decreased compliance, and neck vein distension following an abrupt decrease in end tidal CO_2.

Treatment of Pulmonary Thromboembolism

Treatment is supportive and includes optimizing oxygenation, relieving work of breathing and

maximizing right ventricular preload and increasing forward flow with inotropic support. Current pulmonary vasodilators are non-specific and reduce systemic vascular resistance as well. There may be a role for aerosolized prostacyclin or inhaled nitric oxide in the future. Heparinization remains the mainstay of treatment followed by warfarin. LMWH has also proven efficiency in management of DVT and embolization. Vena caval filters are recommended when there is a contraindication to anticoagulation. There is a definite role for thrombolysis in massive venous thromboembolism, but its role is limited in the perioperative patient.

Venous Air Embolism

First described in 1667 when Francis Redi noted that animals died when air was blown into an open vein. Though it is most commonly associated with neurosurgical procedures in the sitting position, it can occur when there is a significant gradient between the right atrium and an open vein. A gravitational gradient as little as 5 cm is sufficient to allow entrainment of air. Factors modifying air entrainment include:

A. Body position

B. Depth of ventilation

C. Volume of air entering the vessel

D. Rate of gaseous entry

E. Central venous pressure

Diagnosis of VAE

A. Earliest clinical evidence of VAE obtained by TEE. It may be oversensitive.

B. Transthoracic Doppler. Noninvasive. Detects as little as 0.25 ml of air.

C. Capnography. Does not indicate when embolism ceases

D. End tidal N_2. Detects an entry as slow as 1 ml/kg/min.

E. Aspiration of air via CVP multi-orifice catheter

Treatment

Limit further air entrainment by flooding the field and lowering the point of air entry to below the level of the heart. Left lateral Trendelenburg position is controversial though previously recommended to move air out of the RV outflow tract. Nitrous oxide should be discontinued if in use. Jugular venous pressure increases the venous pressure in the head till the site of air entry can be localized and packed. Use of PEEP in this situation is of questionable benefit.

Fat Embolism

Fat embolism occurs when movement or distortion of bone fragments or reaming of the medullary cavity increases the pressure within this cavity and force entry of marrow fat into torn venous channels. This causes occlusion of multiple blood vessels with fat globules that are too large to pass through capillaries. In addition to mechanical occlusion, the hydrolysis of neutral fat to free fatty acids causes damage to endothelium and pneumocytes producing a picture indistinguishable from ARDS. Another feature of fat embolism syndrome (FES) is occlusion of vessels in the brain, kidney, and myocardium through presence of fat globules through pulmonary capillaries or intracardiac shunting.

Diagnosis

Laboratory tests that demonstrate the pressure of fat globules in the circulation of urine do not correlate well to the presence of or severity of the FES. The criteria used for diagnosis are (Gurd and Wilson)

Major Feature (at least 1)

Petechial rash

Respiratory insufficiency

Cerebral involvement

Minor Features (4 of 5)

Pyrexia

Tachycardia

Retinal changes

Jaundice

Renal changes

Fat macroglobulinemia

In conjunction with these criteria PaO_2<60 mmHg and $PaCO_2$>55 mmHg, pHa<7.3 or tachypnea >35 beats/min is used (Lindeque).

Prevention and Treatment

Early immobilization and fixation of long bone fractures is the mainstay of prevention of FES. Treatment is supportive. Use of alcohol infusion, high dose corticosteroids, and aprotinin cannot be recommended at this stage.

Amniotic Fluid Embolism (AFE)

Reported incidence is between 1 in 8000 and 1 in 80,000 pregnancies. The mortality rate is about 60-70%. Meconium in the amniotic fluid at the time of embolism and preexistent fetal death appear to be risk factors for severe AFE syndrome. Analysis of a large series by Clark, et al and Morgan showed no association between tumultuous labour and use of oxytocin, increased fetal size, or route of delivery.

Pathophysiology

Amniotic fluid contains fat from vernix caseosa, squames from fetal skin, mucin and trophoblastic cells. The pathogenesis of the AFE results only partly from mechanical obstruction of the pulmonary vasculature. The predominant efforts seem to be mediated by leukotrienes, thromboxane and brady kinin. The first reaction to APE is sudden severe transient pulmonary hypertension with hypoxemia followed by an increase in systemic vascular resistance. Echocardiography has confirmed global cardiac dysfunction with severe pulmonary hypertension. Coagulopathy develops in half the patients with AFE. Shnider and Moja noted the AFE was characterized by four cardinal signs:

A. Respiratory distress

B. Cyanosis

C. Cardiovascular collapse

D. Coma

Uncontrolled bleeding may also occur. Diagnosis depends on the above signs and demonstration of fetal elements in the maternal pulmonary circulation. It should be noted that small amounts of fetal debris might be found in the pulmonary circulation of asymptomatic parturients.

Treatment

Treatment is supportive. Immediate delivery of the fetus should be strongly considered.

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Airway Emergencies in Paediatrics

Dr. Rebecca Jacob

Prof. and Head, Department of Anaesthesiology, CMC, Vellore

In this talk I will deal with sudden and often life threatening events affecting natural and artificial airways in Paediatrics and will review their identification, assessment and treatment. Of interest will be the special techniques and use of adjunctive equipment for establishing, maintaining and monitoring effective ventilation.

Indication for emergency airway management may be one of the following:

a) Congenital
- Choanal atresia
- Cysts
- Diaphragmatic hernia
- Laryngomalacia
- Mediastinal tumour
- Arytenoid masses

Infection
- Croup - viral
- Epiglottis - Haemophilus influenza
- Laryngo tracheobronchitis
- Tonsillopharyngitis or Retropharyngeal abscess
- Acute uvular oedema

Trauma
- Traumatic upper airway obstruction
- Massive upper airway bleeding
- Foreign body in airway

Obstructive angioneurotic oedema

Chemical ingestion or burns/smoke inhalation

Accidental occlusion of artificial airway. Accidental extubation in an apneic patient.

Assessment of the airway and the need to maintain it should consider

1. The patient unable to adequately protect his airway (coma, lack of gag reflex or unable to cough) with or without other signs of respiratory distress.
2. Does he have respiratory distress with an elevated respiratory rate, high or low ventilatory volume, and signs of sympathetic nervous system hyperactivity.
3. Does he have a partially obstructed airway, signs of which include ineffective patient efforts to ventilate, paradoxical respiration, stridor, use of accessory muscles, patient pointing to his neck, choking motions, cyanosis and distress. Signs of lower airway obstruction may include the above and wheezing.

   The classical sign of airway obstruction is stridor. Usually inspiratory stridor indicates pathology above the larynx and expiratory stridor pathology below the larynx. Problems with the larynx may cause both inspiratory and expiratory stridor.
4. Does he have complete airway observation? Respiratory efforts with no breath sounds or suggestion of air movements.
5. Is he apneic - with no respiratory efforts? Is it associated with cardiac arrest?
6. Do the laboratory findings show hypoxemia, hypercarbia and/or acidosis.

In all these cases care must be taken to provide optimal oxygenation and attempts to secure the airway must be made as early as possible. It may be necessary to gently introduce an inhalation agent to induce sleep without producing apnoea. This process is often very slow due to obstruction to fresh gas flow. If this is possible establish an intravenous access once the child

is asleep. If not, establish the intravenous line while he is awake. Always use a pulse oximeter during induction and an ECG wherever possible. Antisialagogues like glycopyrrolate and atropine (also useful to have diluted and ready in case of bradycardia) must be considered.

Mask ventilation may prove to be difficult. Intubate whenever possible and check the position of tube. Ensure adequate air entry bilaterally. Problems which may be encountered with endotracheal intubation include kinking or biting of the tube, occlusion of tube or inadvertent extubation. Other problems include bronchospasm, laryngospasm, aspiration, dysrhythmia, bleeding and oedema.

Problems with emergency ventilation

- inadequate oxygen delivery
- hypo or hyper ventilation
- gastric insufflation
- baro trauma
- hypotension (due to increased intra thoracic pressure)
- interruption of ventilation for intubation
- Failure to exhale during TTJV - due to upper airway observation.

Timely intervention to maintain the patients airway can improve outcome in terms of survival and level of function. Various clinical scenarios will be presented and options for airway management including mask ventilation, the use of laryngeal mask airways, endotracheal or nasotracheal intubation, trans tracheal jet ventilation, circothyrotomy puncture and tracheostomy will be discussed. The importance of monitoring will be stressed.

Non Traumatic Chest Pain
An Emergency Medicine Approach

Dr. Prem Pais

Professor and Head, Dept. of Medicine, In-charge, Medical Intensive Care Unit,

St. John's Medical College Hospital, Bangalore

Non-traumatic chest pain is a very common symptom for patients presenting to an emergency medicine department (EMD). The patient and their families are frightened and the managing doctor is anxious because of the common belief that chest pain heralds a "heart attack". This anxiety is reasonable and the prime responsibility of the EMD physician in such a situation is to rule out an acute ischaemic cardiac event.

We know that most such patients will have non cardiac chest pain and many of those with ischaemic heart pain will be at low risk of death or "events" at least in the short term but others will be at high risk. Therefore the objectives in managing patients with non - traumatic chest pain presenting to the EMD should be

1. Making a quick assessment to arrive at a likely diagnosis
2. Triaging the patients
3. Initiating early treatment where required

These objectives should be achieved with a sense of urgency.

Delayed hospitalisation: In most emergencies, time is of the essence. This is especially so in ischaemic cardiac emergencies where benefits of reperfusion are inversely proportional to the time to start of treatment. Over half the deaths in AMI due to VF occur in the Ist hour after onset of symptoms.

What are the factors causing delay? The biggest delay is between onset of symptoms and the decision to go to hospital. Thus public education is of prime importance. The second obstacle is transport to hospital. This is especially a problem in India with bad road, congested traffic and poor ambulance services. A prospective study we conducted in 1998 of 1072 patients with AMI across 14 hospitals in South India showed a mean time to presentation to hospital of 11 hours after onset of chest pain. Only 6% of patients used an ambulance to travel to hospital. 80% came by private transport (car, jeeps, autorickshaws), 9% came by public bus and 3% on two wheelers. The third source of delay is in initiation of treatment. While in developed countries treatment is started by trained paramedics in the ambulance or even at the patient's home, ambulances in India by and large merely serve to transport the patient. Can we at least minimize future delay in the EMD.

Causes of Chest Pain: some important causes of chest pain are shown in the table below. Those with urgency are underlined.

I. Cardiac

- Acute Myocardial Infarction
- Acute Ischaemic Syndrome
- Pericarditis

II. Vascular

- Aortic Dissection
- Pulmonary Embolism

III. Pulmonary

- Pneumothorax
- Pneumonia
- Pleurisy

IV. Gastro-intestinal

- Oesophageal reflux
- Oesophageal spasm
- Mallory Weiss syndrome
- Pancreatico-biliary disease
- Perforation

V. Musculo-skeletal and others

- Cervical disc disease
- Costochondritis
- Herpes zoster
- Emotional

Interpreting diagnostic tests: Before discussing diagnostic strategies remember that mechanically taking a test or sign to be positive or negative can be erroneous. One must always exercise one's clinical skill and experience to first assess how likely it is that the patient has the disease being tested for. Strangely the diagnostic reliability of a test is directly proportional to the likelihood that the patient has the condition. In conditions where a patient is not likely to have a disease, tests for that disease become less reliable. Thus while in a 55 year old male hypertensive and diabetic with chest pain an ECG is reliable in a 25 year old healthy woman with the same symptom a treadmill test may have a high false positive rate.

Diagnostic characteristics: Consider the diagnostic characteristics of some life threatening emergencies presenting with chest pain. In all cases characteristics of patient and risk factors must be an important factor in making a diagnosis.

I Acute Myocardial Infarction

- Severe retrosternal chest pain lasting 1/2 hour or more with associated symptoms such as sweating
- ECG - characteristic, but may be non diagnostic
- Enzymes - CK, MB, Troponins
- 3-4 hour window period

Acute Ischaemic Syndromes

- Milder, shorter pain
- Variable ECG and enzymes

50% of those ultimately diagnosed to have AMI and 65% of those with AIS have a non-diagnostic initial ECG.

II Dissecting aneurysm

- Abrupt onset of severe central chest pain radiating to the back
- Unequal pulses and BP
- Widened aortic arch on CXR
- Variable ECG

Pulmonary embolism

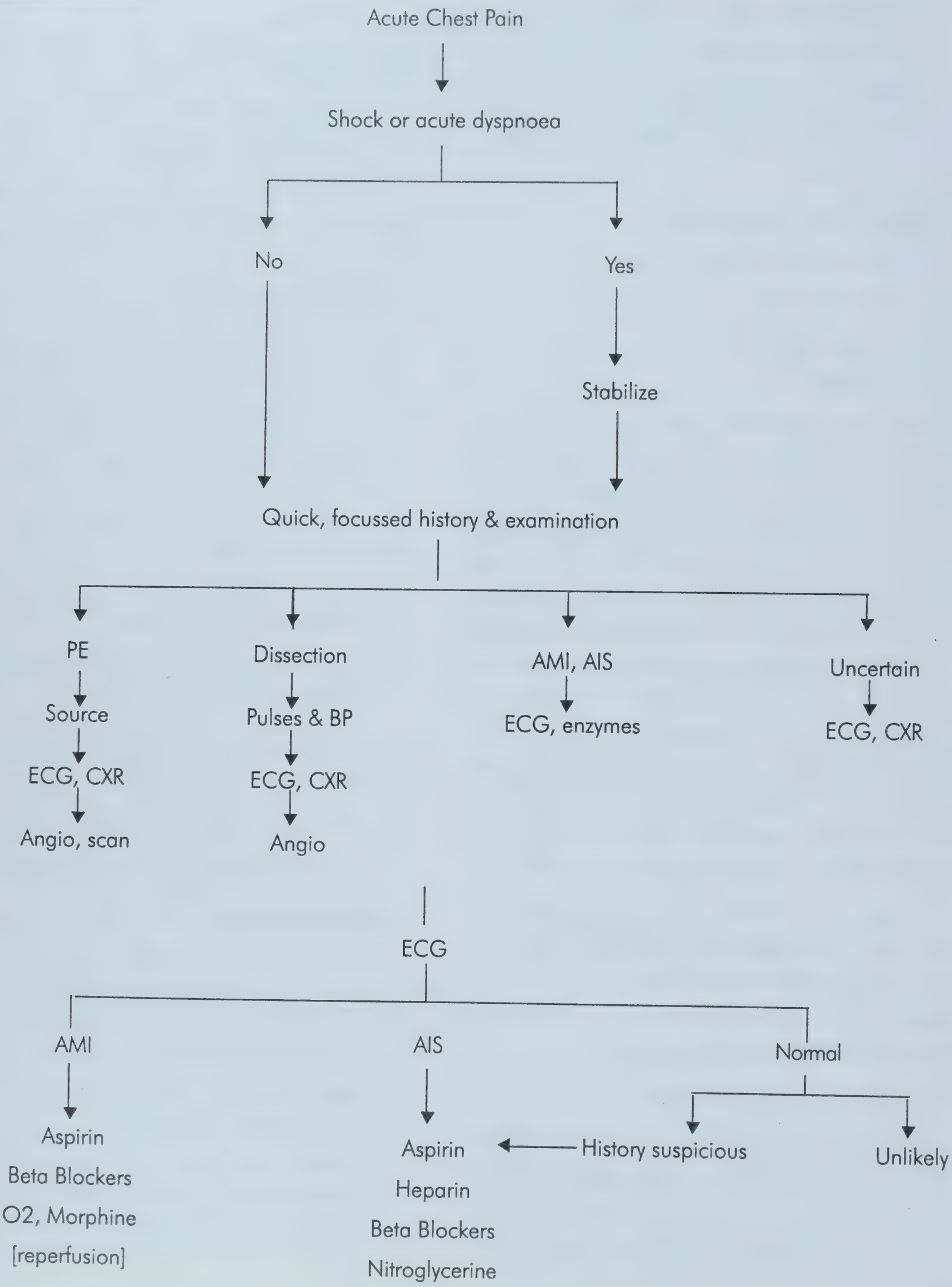
- Large PE can mimic AMI - central chest pain, hypotension & dyspnoea
- Smaller ones present with pleuritic pain haemoptysis and some dyspnoea
- ABG - picture of Type I respiratory failure
- Perfusion ± ventilation scan helpful
- Pulmonary angiogram is the gold standard
- A high index of suspicion is essential.

III Pneumothorax

- Acute chest pain with dyspnoea
- Characteristic signs
- Diagnostic chest xray

IV GI problems - Mallory Weiss Syndrome, cholecystitis, pancreatitis or perforation may present with chest pain but can usually be identified by a good history and careful abdominal examination.

Diagnostic Flow Chart:



Chest Pain Units: Most patients presenting to an EMD with chest pain have non cardiac and non life threatening conditions or are at low risk of adverse events. At the same time, patients at high risk may present with non diagnostic clinical or ECG findings. This has lead to the concept of "chest pain units". In such units patients can be observed for 6-12 hours, further tests done and a more confident decision taken for formal admission or discharge. Therapy can also be initiated in such units. Such tests usually include monitoring ECG, enzymes and at times chest xray. Some units also do a stress test before discharging a patient.

Cardiac Enzymes, some issues:

- CK rises after skeletal muscle injury eg. IM injection
- Troponins may have prognostic value in AIS. Among patients with AIS, those with raised troponin are at higher risk and benefit more from low molecular weight heparin and GPIIB/IIIA receptor blockers (FRISC, CAPTURE). Those with normal troponin after 6 hours have low risk and could be discharged.
- Troponins are normally not detectable in blood and even a small rise is significant.
- Rise of troponins take 4-6 hours

Therapy in EMD: Apart from inserting chest tubes in pneumothorax, stabilizing an unstable patient, giving oxygen and relieving pain, what other therapy can be started in the EMD? In suspected AMI or AIS it would be prudent to give aspirin and probably beta blockers as well (in the absence of contraindications). In AIS heparin and nitroglycerin could be started safely. It should also be possible to work out a mechanism of starting thrombolytic agents without delay but this would depend on institutional policies.

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Cardiopulmonary Resuscitation: The Indian Experience

Ram E. Rajagopalan
Consultant & Head, Department of Crtical Care Medicine
Sundaram Medical Foundation, Chennai 600 040

Despite the lapse of over a quarter of a century since the development and application of standardised protocols for cardiopulmonary resuscitation (CPR) in humans, its popularity in Indian hospitals and in pre-hospital care has been limited by several factors. Inadequacies in the infrastructure, lack of training and the unavailability of equipment raise concerns about the appropriateness of CPR in India (1). It is often presumed, quite logically, that outcomes in India will be poor. Consequently, CPR is seldom performed on victims of cardiorespiratory arrest, especially when it has occurred out of the hospital. Until recently, however, there was no data available to support or refute these claims. Recent studies, though based on small groups of patients, allow us to make some evidence-based judgements.

Survival after in-hospital cardiorespiratory arrest

Reports are available from an urban community hospital (2) and a tertiary care centre (3) indicating that CPR done on in-patient arrests in these hospitals were associated with outcomes that may be comparable to that reported from developed nations (4) . The return of spontaneous circulation (ROSC) was seen in 42-72% of all cases in whom resuscitation was attempted, resulting in about 18% of patients being discharged home alive.

Multivariate analysis of the predictors of outcome reveal nothing that is significantly new. While there appears to be an unexplained increase in the survival of women, the most influential factors appear to be the presence of a "shockable" rhythm (ventricular fibrillation or ventricular tachycardia) at presentation (2,3) and the occurrence of the arrest in hospital (2). The overall implications of these studies is that even in Indian hospitals, CPR can be an effective procedure that results in the live discharge of one patient for every 5.5 attempts made.

However, with large variations that exist in the infrastructure and monetary resources available to the resuscitator, extrapolation of these results to all urban, let alone rural hospitals, is difficult (1). For example, while the studies show that defibrillation is an important factor in successful Advanced Life Support (ALS), only 27% of a group of hospital-based physicians surveyed in Chennai reported that they had access to a defibrillator at all times (5). While it is currently possible to achieve good outcomes in select centres in the country (2,3), widespread success in the application of ALS would be greatly dependent on major improvements of the infrastructure.

Should we perform CPR on out-of hospital arrests?

No city in India has an adequate network of rapid-response paramedic teams that can provide pre-hospital care for cardiac arrests occurring out of the hospital. With considerable delays in transportation, absence of pre-hospital basic CPR and delayed access to defibrillation outcomes may be predicted to be poorer than in arrests in the hospital. Data on this aspect of CPR in India is even more scarce.

We have reported outcomes from our own emergency department (ED) in which the policy is to attempt resuscitation on all out-of-hospital arrests irrespective of the quality of pre-hospital support (6).

None of the 68 victims in our study were transported by ALS trained physicians or paramedics and only 4% received bystander CPR (BLS) prior to arrival at the ED. 15% had either ventricular tachycardia or fibrillation as the presenting rhythm. Though ROSC was similar to that seen in the in-hospital arrests (42%), only 4 patients (5.9%; 95% CI 1.6-14.4) survived to be discharged alive from the hospital.

While these outcomes are significantly poorer than that seen after in-hospital CPR, certain subgroups appear to fare reasonably well even under these conditions. Thus, if ventricular tachycardia or fibrillation was the rhythm at the initial presentation, there was a trend towards improved survival. 20% of patients with these rhythms were alive at discharge in contrast to 3.4% with PEA or asystole (RR = 5.8; 95% CI 0.92-36.6). Also, despite its infrequency, the performance of bystander CPR resulted in better survival (RR = 7.22; 95% CI 1.03-50.5). No patient undergoing-CPR for greater than 20 minutes survived to be discharged, while 15% of CPRs less than 20 minutes resulted in a live discharge (P = 0.02).

It is therefore our impression that, in an urban community setting, there are clear predictors of successful resuscitation after a pre-hospital arrest. We feel that a policy of "no CPR" in all out-of-hospital events is not justifiable even on the-basis of this preliminary data. It is imperative to identify ventricular fibrillation or tachycardia using "quick look" paddles, especially if bystander CPR had been performed. Resuscitation of such individuals could lead to the discharge of one live patient for every five attempts. Education of the public about BLS will also be a cost-effective policy to improve survival under these conditions.

Education in ALS

Given all the limitations that exist in performing CPR in India, we feel that the first step in improving the quality lies in initiating a wide based education programme. We feel that the average Indian physician lacks both the cognitive and procedural skills that are necessary for the efficient administration of advanced CPR. When we surveyed 200 hospital-based physicians, who performed CPR, we found a tot of discordance between their opinions and the guidelines of the American Heart Association and the European Resuscitation Council that were current at the time of the study (5). Although we could not test performance, the self-reported rates of competence in ALS procedures such as airway maintenance were poor. Their understanding of CPR-related pharmacology also left much to be desired. This study made us conclude that there was an urgent need for the education of medical professionals in ALS. At present, ALS courses are rare in India and are generally not a part of graduate or postgraduate medical curricula.

BLS Education & Public attitudes to CPR

The studies on both in-hospital and pre-hospital CPR show us that bystander CPR plays a crucial role in emergency cardiac care (2). However, in both situations BLS was performed far too infrequently, being used in only 25% of eligible in-hospital arrests and 4% of out-of-hospital events (2,6).

We realise that the infrequency of bystander CPR is not just a reflection of public apathy, but may be due to the scarcity of BLS education. Public attitudes to CPR in India do not appear to be different from that reported elsewhere. In a survey of the lay participants in our BLS courses (7), we found that there is a general willingness to perform chest compressions, irrespective of the victim's age or sex or of the rescuer's relationship to the victim. However, these factors would influence the frequency with which mouth-to-mouth-respiration was given. Fear of contracting disease and concerns about public reactions also seem to contribute to this disinclination. The successful application of BLS by

bystanders in India will greatly depend on how these concerns about mouth-to-mouth respiration are addressed.

Conclusion

While there are numerous hurdles to the widespread delivery of CPR in India, studies from dedicated ALS teams in urban hospitals indicate that outcomes can be significantly improved to levels that are comparable with that seen in the developed nations. With inadequate knowledge being the first hurdle, significant strides can only be made by educating medical, paramedical and lay rescuers. Simple approaches to improving the infrastructure such as increasing the availability of defibrillators will have more far-reaching consequences than large expenditures on sophisticated pre-hospital emergency rescue and transport networks.

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Hypertensive Emergencies

Ram E. Rajagopalan, MBBS, AB (Int. Med. & Crit. Care)
Consultant & Head, Department of Critical Care Medicine
Sundaram Medical Foundation, Chennai 600 040

The management of acute severe hypertension associated with life-threatening end organ dysfunction is a therapeutic challenge in emergency medicine. Even with increasing knowledge about hypertension amongst the public and despite the greater choice of treatment options, such patients are still commonly seen in the emergency department (ED). As has been traditionally emphasised, there is a crucial difference between the *hypertensive urgency* - in which blood pressure can be lowered gradually, usually with oral medication over several hours - and the *hypertensive emergency* in which aggressive early therapy, often using parenteral medication is needed to reduce the pressure within minutes to a few hours after presentation. This discussion will focus on the latter group.

An urgency cannot be distinguished from an emergency on the basis of the severity of hypertension. Thus even a "mild" elevation of blood pressure to say 160/100 mmHg may be associated with encephalopathy and convulsions in a pregnant woman with eclampsia. In the converse of this situation, very severe elevations are not uncommon in asymptomatic hypertensives without target organ damage who can be safely treated without admission to the hospital. It is the presence of acute and progressive target organ dysfunction that makes the distinction. Thus the classical hypertensive emergencies are

a. Hypertensive encephalopathy
b. Hypertensive left ventricular failure
c. Hypertension with acute coronary syndromes including myocardial infarction
d. Hypertension with acute aortic dissection

Unlike in the past, when *accelerated* and *malignant* hypertension were considered different entities, current evidence does not suggest that the latter, which presents with the fundoscopic finding of papilloedema, is prognosticaly different from accelerated hypertension. However, under these circumstances too, the development of other end-organ damage gravely affects prognosis and needs to be tackled as an emergency.

Physiological concerns

While treating a hypertensive emergency a few basic aspects of the physiology of hypertension should be understood. Primarily there is the concept of *autoregulation*, a mechanism that protects an organ from ischaemia when there is a fall in blood pressure. It also prevents hyperaemia and oedema formation when there is a significant rise in the pressure. Thus in normal individuals, the blood flow to organs such as the brain remains unchanged even when the mean arterial pressure fluctuates between 60 and 150 mmHg. Neurological manifestations occur only when these limits are exceeded.

In the chronic hypertensive both the upper and lower limits are reset at a higher value and reduction of blood pressure even to what may be "normal" may compromise flow to the brain. Recognising this is very crucial to patient management, as aggressive attempts to "normalise" blood pressure can result in renal or neurological deterioration. It has been noted that the lower limit of autoregulation in both normotensive and hypertensive patients is about 25% below the resting mean arterial pressure. Thus *reducing the blood*

pressure by about 25% is a safe goal in the management of hypertensive emergencies. Drugs that produce uncontrollable drops in BP should be used with great caution, as "overshoot hypotension" could affect outcomes adversely.

Exceptions to this rule are encountered while dealing *with cardiac conditions* (LV failure, unstable coronary syndromes and aortic dissection), where *a greater reduction of BP will reduce afterload and myocardial oxygen demand.* In these cases, dropping blood pressure by more than 25% may be beneficial but should be done with close observation of neurological status and urine output.

Finally, the *acutely hypertensive individual may often be dehydrated because of pressure induced natriuresis.* This has two major implications on treatment. Primarily, *diuretics, that are effective agents in chronic hypertension, have no role in the initial management of hypertensive crises.* On the other hand, the administration of vasodilators may further decrease vascular filling in the dehydrated patient. Declining urine output under these conditions may require fluid loading, which must be done carefully, preferably using invasive pressure monitoring.

The initial approach

The initial approach to a hypertensive emergency is clearly influenced by the presenting symptoms. Thus the patient may present in an unconscious state (encephalopathy or aortic dissection), in acute respiratory distress (LV failure, unstable coronary syndromes) or complaining of chest pain (MI, unstable angina or aortic dissection). As in all emergencies *the "ABC" approach* intending to stabilise the airway and breathing and establish the presence of circulation, is followed by an evaluation of vital signs including blood pressure measurement.

While supportive care (the provision of oxygen and establishing an IV access) is being provided it is essential to consider a differential diagnosis that includes a number of conditions that can mimic a hypertensive crisis. In this situation it must be realised that *hypertension may, as often, be the consequence of a medical crisis, as the cause of it.*

Thus a normotensive individual, who is hypoxic and in distress from pulmonary oedema induced by coronary insufficiency, may have a high blood pressure from the catecholamine surge associated with the stress of the acute crisis. Likewise, an unconscious patient with an intracranial haemorrhage or space-occupying lesion may have the classical Cushing reflex (bradycardia & hypertension), which is an attempt to maintain cerebral blood flow during the acute crises. In either situation aggressive attempts at lowering the pressure may be deleterious to the patient. Also, in acute catastrophic neurological events, cerebral autoregulation is lost, making the brain extremely vulnerable to drops in blood pressure. *Given that such "reactive" rises in blood pressure are far more common than genuine hypertensive crises, a conservative approach to the initial reduction of BP should be used.* Drugs such as oral nifedipine that do not allow a titratable decrease of pressure should be avoided as initial treatment (see below).

Selection of drugs

Several factors influence the choice of drugs used in a hypertensive emergency:

a. As a rapid reduction of pressure is desired, parenteral agents are usually preferred. This is not an absolute rule, as limitations exist in India in the availability of parenteral drugs, the cost of medication and the cost of monitoring, compelling one to use oral agents, of which nifedipine has acquired a prominent role.

b. The properties of the drug must to be considered in relation to the specific indication for its use. Thus

vasodilation with nitroprusside in aortic dissection will lower blood pressure but increase shear forces, which can be curtailed by combining it with a beta-blocker. Vasodilators like hydralazine and nitroprusside may cause rising intracranial pressure by dilating cerebral vessels and are to be used cautiously in hypertensive encepahlopathy. Beta adrenergic receptor blockers are contraindicated in the acute management of LV failure. The table below provides guidelines for the treatment of hypertensive emergencies.

Emergency	Agent of Choice (IV)	Agents to be avoided	Comments
Hypertensive Encephalopathy	Nitroprusside * or Trimethaphan** or Labetalol**	Methyldopa / Reserpine (sedation) Diazoxide / hydralazine (increase cerebral blood flow)	* needs invasive arterial pressure **limited availability in India @ initiate at venodilatory doses (10-40 ug / min) and advance as needed to arterial dilatory doses (1.5 to 2 ug/kg/min). With non-invasive monitoring, small boluses of 1-2 mg can be used during titration
L V Failure	Nitroglycerine@ or Nitroprusside* or Enalaprilat**	Beta blockers - esmolol, labetalol, propranolol etc.- (reduce cardiac output)	
Unstable Coronary Syndromes	Nitroglycerine@ or Nitroprusside* or Beta Blockers	Hydralazine / diazoxide (increase myocardial work/ O_2 consumed)	
Dissecting aortic aneurysm	Trimethaphan** or Nitroprusside with beta blocker*	Hydralazine / diazoxide (increase myocardial work / O_2 consumed)	

c. The extent of monitoring required may also pose a problem in the use of some drugs. As a rule, we feel that all hypertensive emergencies require frequent monitoring of blood pressure with the use of automated non-invasive blood pressure monitors. If nitroprusside is being considered for treatment invasive arterial pressures are mandatory. The limitations in the availability of the latter make this effective anti-hypertensive agent unpopular.

d. Finally, given many of these limitations, it may be necessary to use oral nifedipine. This drug is very effective in reducing blood pressure. However, the inability to control the extent of decline in blood pressure is often cited as a reason to avoid this drug. Though overshoot hypotension is reported it is very rare with doses of 10 mg or less. We feel that the safety profile can be improved if simple precautions are taken in administering the drug:

i. Avoid its use in "reactive" hypertension. Provide reassurance, oxygen, pain relief etc. and consider initiating therapy with a titratable drug such as iv nitroglycerine. If the blood pressure remains high after these measures use nifedipine.

ii. We prefer a smaller dose (5 mg), especially in the elderly, who may have significant cerebrovascular disease and may tolerate drops in blood pressure poorly.

iii. Contrary to popular belief the drug has its best effect if the liquid-filled capsule is bitten and swallowed rather than when the liquid is placed sublingually. In fact, if a slow effect is desired, some recommend the use of the sublingual route.

iv. Avoid its use in acute myocardial infarction. In patients with unstable coronary syndromes - with or without left ventricular failure - we find repeated boluses of nitroglycerine extremely effective in lowering blood pressure (see table). The lower blood pressure can be sustained with appropriate infusion of the drug.

Conclusion

The challenge in management of hypertensive emergencies in India lies in the limitations imposed on the physician in terms of availability of drugs, cost of treatment and availability of monitoring. Broad principles can be developed for treatment, but these rules have to be modified to suit the infrastructure available and the expenditure that the patient can afford.

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Hypertensive Emergencies in Pregnancy

Dr. Nirmala Soundararajan
Fernandez Maternity Hospital, Hyderabad

Hypertensive disorders in pregnacy remain one of the leading causes of maternal mortality worldwide accounting for 15-20% of maternal deaths in developing as well as developed nations.

The term "hypertensive crisis" is loosely defined as a severe elevation in blood pressure. In 1986, Ferguson and Vlasses divided hypertensive crises into **hypertensive emergencies** and **hypertensive urgencies**. Based upon this concept, there is no arbitrary level of blood pressure that distinguishes a hypertensive emergency from urgency. Emergencies are characterised by progressive end-organ dysfunction and require correction of blood pressure within one hour. Evidence of end-organ dysfunction in the history, on physical examination or by laboratory evaluation differentiates hypertensive emergency from urgency. Examples of hypertensive emergency include hypertensive encephalopathy, acute left ventricular failure with pulmonary edema, aortic dissection and eclampsia. In contrast, hypertensive urgencies are characterised by marked hypertension but do not have end-organ dysfunction and require control within 24 hours; examples include accelerated hypertension without impending complications and hypertensive patients who require emergency surgery.

The practical issues that need to be addressed when a hypertensive crisis is encountered are:

a. Is it a hypertensive emergency?
b. What is the level of blood pessure that should be achieved by therapy?
c. Is a parenteral agent preferable?
d. Is a short duration of action preferable?
e. Is there a danger due to overshoot hypotension?

Issue 1

Is this a hypertensive emergency?

Evidence of end-organ dysfunction can be obtained:

- in the history : chest pain, neurologic changes, shortness of breath
- on physical examination : new retinopathy, papilloedema, neurologic deficits, rales, S_3, or pulse deficits, or
- by laboratory evaluation : ischaemic changes on the ECG, pulmonary edema on chest radiograph, increased serum creatinine, hematuria, hemolysis on the blood smear.

Issue 2

What is the level of blood pressure that should be achieved by therapy ?

The objective of treating severe hypertension is:

a. To avoid loss of cerebral autoregulation and subsequent maternal cerebrovascular accidents.
b. To prevent congestive heart failure without compromising cerebral perfusion or jeopardising uteroplacental blood flow.

Ideally, the systolic blood pressure is kept between 140 mmHg and 150 mmHg and the diastolic between 90 mmHg and 100 mmHg.

Issue 3

Is a parenteral agent preferable?

A parenteral agent is preferable in emergency situations. Reduction of blood pressure to desired values need to to be achieved preferably within one hour and definitely within 4 hours.

Parenteral agents are also necessary if the patient has altered sensorium, is unconscious, is scheduled for emergency surgery, has paralytic ileus, or for some reason is unfit for oral therapy.

Issue 4

Is a short duration of action preferable?

Parenteral agents with a short duration of action are preferable in emergencies because drug titration is easier and overshoot hypotension can be prevented easily by reduction of dose or discontinuation of drug.

Issue 5

Is there a danger due to overshoot hypotension?

Uteroplacental circulation is sensitive to excessive reduction in perfusion pressure. Resultant blood pressure must be low enough to provide safety for the mother without making the intrauterine environment hostile for the fetus. Normal blood pressure levels are avoided antepartum because of the risk of inducing dangerous decrements in uteroplacental perfusion. It is possible to induce transient late decelerations of fetal heart rate when the blood pressure is lowered acutely. This is more likely when there is fetal growth restriction.

Etiology of Hypertensive Emergencies

Hypertension is the most common medical disorder that complicates 5-10% of all pregnancies. In our hospital 125/2804 deliveries i.e. 4.46% were complicated by hypertension.

Pre-eclampsia is a uniquely human complication of pregnancy and of unknown etiology. Pre-eclampsia is the cause in 70% of patients with acute onset hypertension in pregnancy. Pre-eclampsia is typically characterised by proteinuria and generalized edema in addition to elevated blood pressure. Atypical pre-eclampsia can be present with various of these three components in addition to hemolysis; thrombocytopenia and evidence of hepatic dysfunction as the HELLP syndrome. All patients with atypical pre-eclampsia are considered to have severe disease.

Eclampsia and Pre-eclampsia are the most important obstetric causes of maternal morbidity and mortality worldwide. Acute life-threatening complications can arise from the functional derangements of multiple organ systems. Abruptio placentae is the most frequent complication whereas intracerebral haemorrhage is the most serious.

The other causes of new onset hypertension in pregnant patients include chronic hypertension, chronic hypertension with superimposed pre-eclampsia and other rare causes such as pheochromocytoma, thyrotoxicosis, active lupus erythematosus and substance abuse.

"Assessment of the Hypertensive Patient"

Symptoms

Headaches, visual disturbances, flushing, dyspnoea, chest pain, nausea and vomiting.

Vital Signs

Blood pressure, pulse rate, respiration, weight, fluid intake and output.

Physical Examination

Eyes: jaundice, papilloedema, double vision

CNS: localising signs, mental status, headache

Peripheral nervous system : Localizing findings, reflexes.

Chest: Cardiac rhythm, murmurs, pericardial effusion, pulmonary edema.

Abdomen: Epigastric pain, uterine enlargement, ascites, edema of the abdominal wall and pre sacral edema.

Extremities: Dependent edema, normal reflexes, equal pulses, tenderness suggesting possible thrombosis.

Pelvic Examination

Laboratory

Imaging: Baseline chest Radiography

Baseline pelvic ultrasound of the uterus

Chemical:

Complete blood count with differential count and peripheral smear.

Platelet count

Baseline coagulation profile: PT, APTT fibrinogen, FDP

Hepatic: LDH, AST, ALT, bilirubin (direct and indirect)

Urine Examination

Urine dipstick for protein and 24 hours urine collection for protein and creatinine clearance

Renal

Creatinine, BUN, S.electrolytes, S.Uric Acid.

Miscellaneous

Arterial blood gas analysis and blood glucose

Management

The corner stone of management in pre-eclampsia is **"stabilize and deliver"** and this principle has four aspects.

a. Seizure prophylaxis

b. Fluid therapy

c. Anti hypertensive therapy

d. Anaesthetic Management

At this point, it must be reiterated that *comprehensive care is more important than drug choice.*

A. Seizure prophylaxis

The protocol is based on Pritchard and Pritchard Magnesium Sulphate regime and is as follows:

20 ml of a 20% solution is given IV over a period of 5 minutes slowly. This dose is equivalent to 4 gm. Simultaneously 20 ml (10 ml in each buttock) of a 50% solution is given intra-muscularly. This is equivalent to 10 gm of magnesium sulphate. Therefore total loading dose is 14 gm.

Maintenance Dose

A 50% solution of 10 ml (5 gm) is given IM on alternate buttock at intervals of 6 hours for 48 hours or for 24 hours after delivery.

B. Fluid therapy

Pre-eclampsia and eclampsia are characterized by the lack of normal pregnancy induced hyper volemia. Fluid intake is restricted unless there is excessive blood loss or oliguria. Lactated Ringer's solution at the rate of 100 ml/hr is given.

C. Anti hypertensive therapy

All the authorities on the management of hypertensive emergencies in pregnancy accept parenteral hydralazine as the drug of choice. However, hydralazine is not available to us. Labetalol is said to be the next most commonly used drug. Unfortunately Labetalol too, is not available in India.

The parenteral agents available to us include sodium Nitropruside and Nitroglycerine. Sodium Nitropruside is an equal arteriolar and venular vasodilator which has a rapid onset of action and is very potent. Therefore monitoring blood pressure using an indwelling arterial catheter is recommended. Fetal cyanide toxicity is a major concern if SNP is used for more than 30 minutes. Therefore, it is recommended only in refractory hypertension unresponsive to other measures.

Nitroglycerine is primarily a venular vasodilator that can be used for minute to minute titration of blood pressure in pregnancy. It has a rapid onset and short duration of action. The dose varies from 0.25-2 μg/kg/min. and the syringe pump enables easy titration.

Nicardipine - a dihydropyridine calcium channel blocker available abroad for oral and intravenous therapy has promise but there is not much published experience with this agent in human pregnancy.

Oral Nifedipine is still the most popular and widely used drug for acute anti hypertensive therapy in pregnancy and can be used as a first line drug. Oral Clonidine and Prazosin are used as second line drugs in hypertensive urgencies. Diuretics are indicated in pregnancy only when pulmonary oedema develops. ACE-inhibitors are contra-indicated in pregnancy.

At this juncture, I would like to quote William C. Mabie (*Clinical Obstetrics & Gynaecology* Vol. 42 No. 3 Sept 1999) who gives a few caveats about antihypertensive therapy.

1. There is great individual variability in response to these drugs, and they do not lower blood pressure predictably, precisely or smoothly.
2. Lowering blood pressure too rapidly or excessively may produce fetal distress, particularly in the setting of intra-uterine growth retardation or an abnormal fetal heart rate tracing.
3. Epidural Anaesthesia will lower the blood pressure by approximately 15%, frequently abrogating the need for anti-hypertensive medication.
4. The gravida with chronic renal insufficiency has hypertension that is much more difficult to control, in part due to volume expansion.
5. Severe hypertension without proteinuria should prompt a urine screen for cocaine.
6. With prolonged unconsciousness, papilloedema, lateralising signs, seizures on magnesium sulphate, or seizures more than 48 hours after delivery, a CT scan should be performed to rule out intracranial haemorrhage.

D. *Epidural Analgesia/Anaesthesia for Labor and Delivery*

Epidural analgesia is the preferred form of pain relief for labor and delivery in pre-eclamptic women for several reasons.

i. Epidural block affords the best pain relief.

ii. Pre-eclamptic women have an exaggerated hypertensive response to pain, which is attenuated by epidural analgesia.

iii. Epidural analgesia reduces the circulating levels of catecholamines and stress-related hormones, which facilitates blood pressure control.

iv. Epidural analgesia may result in improved intervillous blood flow in pre-eclamptic women.

v. Epidural analgesia results in stable cardiac output.

vi. Finally, pre-eclamptic women are at increased risk for caesarean section compared with normotensive parturients, and early administration of epidural analgesia for labour facilitates the subsequent administration of epidural anaesthesia for emergency caesarean section.

Invasive Hemodynamic Monitoring

The ACOG technical bulletin no 175 in December 1992 outlined the indications for invasive hemodynamic monitoring. A consensus statement on the use of the pulmonary artery catheter for various indications was published in 1997 (after a PA catheter consensus conference) in the journal of critical care medicine.

Hypertensive emergencies in pregnancy differ from those in non-pregnant adults. Hypertension in

pregnancy has an undefined etiology but a definite treatment i.e. termination of pregnancy. Therefore clinical management is challenging and decisions regarding timing of delivery have profound lifelong effects on both the mother and the child. Complete recovery, without sequalae, is possible if intervention is timely inspite of the potential for life threatening complications.

As severe pre-eclampsia is characterised by multisystem involvement, it can present challenges which may only be successfully met by facilities and expertise offered on either high dependency units or intensive care units. The management of acute crises is ideally done by a multi speciality team of obstetricians and anaesthetists, although others such as physicians may need to be called upon to help.

I conclude by reiterating that obstetric units supervising high risk deliveries should have an intensive care unit so that both expert obstetric care and high dependency care can be ensured.

Pre Hospital Thrombolytic Therapy - Is it Feasible in India?

Dr. U.B. Khanolkar

Wockhardt Hospital & Heart Institute, Bangalore

Concept of pre-hospital treatment of acute myocardial infarction (AMI) was introduced in the early 60's. The rationale for early treatment for AMI stemmed from the fact that the highest mortality of AMI occurred in the first 1-2 hours of onset of chest pain. Early and appropriate thrombolytic therapy of AMI is expected to restore infarct artery patency, limit infarct size, improve left ventricular function and reduce mortality. However, how early is it possible and safe to thrombolyse? Is pre hospital thrombolysis beneficial, especially in the Indian Subcontinent?

Early animal studies have demonstrated that approximately 64% of myocardium can be salvaged after 40 mins of coronary artery occlusion and only 11% 3 hours after occlusion. International study of infarct survival (ISIS-2) and Gruppo Italians per studio della streptochinasi nell infarcts miocardico (GISSI-1) trials revealed that early treatment resulted in greater reduction of mortality. 56% reduction in mortality was observed in those treated within 1 hour of onset of symptoms in ISIS-2 and a 46% reduction was observed in GISSI-1. However mortality reduction in those treated 3-6 hours after onset of symptoms was only 17%. In GUSTO 1 there was a remarkable reduction in 30 day mortality in those treated within 2 hours of onset of symptoms (5.5%) compared to 4 hours (9%). This benefit was more pronounced in the Myocardial infarction Triage and intervention MITI pre-hospital thrombolytic trial. The mortality rate in those treated within 70 mins of onset of symptoms was 1.2% as compared to 8.7% in these treated after 70 mins. Meta-analysis of major randomised trials demonstrated that in the first couple of hours after onset of chest pain the relationship between time of treatment and survival is more exponential. However the relationship becomes more linear after this period. Benefit of thrombolytic therapy was translated as 65, 37, 26 and 29 lives saved per thousand treated patients in 0-1, 1-2, 2-3 and 3-6 hours interval respectively.

A major hurdle limiting the benefit of thrombolytic therapy is failure to treat in the early hours of AMI. The reasons for delay could be :-

(a) failure on part of the patient to recognise and react to symptoms.

(b) Delay in summoning medical assistance after onset of symptoms - probably 81-160 mins

(c) Delay in instituting treatment after hospital arrival - median delay 31-80 mins.

A logical solution to these problems may be pre-hospital thrombolytic therapy. If pre-hospital thrombolytic therapy were to be successfully instituted then:

(a) personnel have to be trained to diagnose AMI and treat the complications of AMI.

(b) thrombolytic agent should be safely administered without undue complications.

Clinical trials have revealed the beneficial effects of pre-hospital thrombolytic therapy. Koven et al published the first study in 1985 wherein 9 patients received pre-hospital streptokinase administered by a physician attached to a Mobile Coronary Care Unit (MCCU) and 44 patients received hospital treatment. This small trial established the feasibility and safety of pre-hospital thrombolytic therapy. MITI was a multicentre study to determine the clinical outcome pre-hospital thrombolytic therapy. Interesting

observation was that median time saved by pre-hospital thrombolysis was 33 minutes. In the Grampian Region Early Anistreplase Trial the time saved was 130 min. In various major pre-hospital thrombolytic therapy studies the time saved by pre-hospital thrombolysis was 33 - 130 min.

Thrombolytic therapy can be administered by physicians, doctors on board MCCU and trained paramedics. Whoever attends to the patients at home should be proficient in performing cardio-pulmonary resuscitation including defibrillation and able to manage the complications of AMI and thrombolytic therapy.

Pre-hospital thrombolytic agent (a) should be easy to administer (b) should have least possible side effects, which could be managed in a non-hospital setting (c) should result in highest early coronary patency rates and (d) should be cost effective. Ease of administration (bolus injection over 5 min) should favour regular use of anistreplase. However, it has to be stored at 2-8^{0}C and it has a relatively short shelf life. Accelerated TPA has been proved to been associated with high 90 mins TIMI 3 flow compared to streptokinase and APSAC. However, it has a relatively cumbersome infusion regimen. Reteplase has been associated with more rapid and complete patency rates without compromising the safety. In addition it can be easily administered either in hospital or pre-hospital setting. However, it is significantly more expensive as compared to streptokinase. Overall streptokinase may be a favoured thrombolytic agent especially in the Indian socio-economic context.

Does pre-hospital thrombolytic therapy improve mortality and improve left ventricular function as compared to hospital therapy? The European Myocardial Infarction Project (EMIP) study was originally designed to recruit 10,000 patients but was terminated due to lack of fund after 5469 patients were enrolled. Pre-hospital thrombolytic therapy was administered by MCCU staff in 163 countries. Pre-hospital thrombolytic therapy resulted in a nonsignificant trend towards lower mortality as compared to hospital thrombolytic therapy (9.7% v/s 11.1% p=0.08). Early pre-hospital thrombolytic therapy improved mortality without significant improvement in left ventricular function in a group of 87 patients treated with r-tPA. Individual trials have not revealed reduction in in-hospital or 30 day mortality in pre-hospital treated group. However pooled data from the major studies show 16.7% reduction in mortality indicating 18 extra lives saved per 1000 patients. Interestingly 42% reduction in mortality is observed in subgroup who receive thrombolytic therapy 90 mins earlier. Hence the social environment, which is most suitable for pre-hospital thrombolytic therapy would be where the travelling distance between home and hospital is most and the MCCU is appropriately developed. Also those patients who are most benefited are who carry a high risk of mortality such as killip class 2-3 and preinfarction angina.

In conclusion, pre hospital therapy tends to improve early and late morbidity and mortality associated with AMI. However, it should be administered by adequately trained MCCU personnel. The incremental cost of the MCCU may be a deterrent factor in pre-hospital thrombolysis of AMI.

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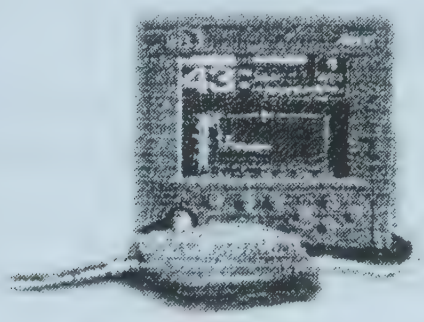
Non Invasive Monitoring During the Care of Patients in Emergency Medicine Departments - A Brief Update

Kumar G. Belani, M.D.

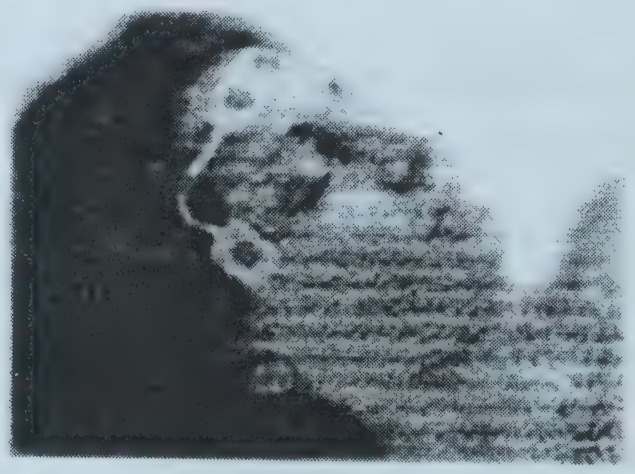
Professor of Anesthesiology, Fairview-University Medical Center, University of Minnesota, Minneapolis, MN 55455, USA

Monitoring of patients' reporting to the Emergency Department is based upon their status. Minimum monitoring includes recording of pulse rate, blood pressure, respiratory rate and core temperature. Body weight is recorded when possible. Depending on the situation, it may be necessary to monitor gas exchange efficiency by pulse oximetry, neurological monitoring by recording level of consciousness and in obstetric patients fetal monitoring when indicated. Patients who are dehydrated or those reporting with multiple trauma will require a urinary catheter to monitor urine output. Some patients might require minor procedures done in the operating room and it would be beneficial to monitor blood pressure continually non-invasively. Such monitoring would also be helpful in patients with coronary ischemia to track blood pressure changes expeditiously. Similarly, it may be necessary to monitor anesthetic and sedative effects on the brain by employing the bispectral index (BIS) monitor.



The BIS Monitor displaying the BIS Index

In a recent study by Song et al. (Anesthesiology 1997; 87:4), the authors found that the use of the BIS monitor helped titrate the anesthetic agents resulting in decreased use of anesthetic agents along with a more rapid recovery profile. Thus BIS could be used to better titrate sedative agent use in Emergency Medical Departments. The system is quite simple to use and requires the application of only 3 electrodes, two on the forehead and one over the temporal region as shown in the picture below:



Non-invasive blood pressure monitoring is usually done by auscultation, oscillometrically or by palpation. Recently, a new method to measure BP frequently non-invasively called the Vasotrac has been introduced (Can J Anaesth. 1999 May; 46 (5 Pt 1): 488-96). The system consists of a wrist module with a pressure transducer. By applying pressure over the radial artery at the distal end of the radius, the pressure transducer assesses the maximum energy transfer of pulsatile beats to determine BP and display a calibrated arterial pressure waveform similar to that obtained directly by a catheter transducer system placed in the radial artery. The system requires 12-15 beats to display the calibrated waveform and arterial BP. The system is as accurate as directly obtained radial artery pressure, has few limitation and may be the answer to continual non-invasive BP monitoring of critically ill patients. In

Emergency rooms this could be of value in patients' with impending myocardial infarction, obstetric patients presenting with eclampsia and requiring quick and precise blood pressure control and patients requiring close monitoring during resuscitation. The Vasotrac has been validated in volunteers, in surgical and critically ill patients and those requiring deliberate hypotension to reduce blood loss.

The Vasotrac Non-Invasive Blood Pressure Monitor displays arterial pulse pressure wave form, systolic, diastolic and mean blood pressure along with pulse rate. The smart sensor is wrapped around the radial artery at the wrist. It applies gentle pressure over the radial artery to measure blood pressure and pulse waveform information. By pressing and retracting repeatedly, BP is displayed continually almost similar to that obtained directly via a radial artery catheter.

Use of non-invasive continual blood pressure monitoring should allow closer monitoring of patients requiring anticoagulation.

In conclusion, reliable non-invasive monitoring of several physiological parameters in patients presenting to Emergency Medical Departments in now becoming increasingly possible.

The table below is an historic chronological list of instruments developed to measure blood pressure indirectly

Method	Comments
• Arterial occlusion by weights	• Cumbersome; not practical
• Counter-pressure occlusion (von Basch, 1876; Potain 1902)	• Fluid filled and air-filled systems; occluding pressure is systolic pressure
• Hydraulic counterpressure on the forearm (Marey 1876)	• Water filled forearm/hand cuff with a window to observe blanching; attached to a recorder; not practical clinically
• Rubber bladder compression of forearm (Hurthle, 1896)	• Systolic pressure was appearance of maximum oscillations and diastolic pressure minimum oscillations
• Air-arm cuffs (Riva-Rocci, 1896 Italy; Hill and Barnard, 1897 England)	• Increased practical application of BP measurement; Cuff-size for oscillometric accuracy (Von Recklinghausen, 1901)
• Korotkoff (1905) introduces auscultatory determination of BP using cuff occlusion methods	• After initial denial, this method became widely accepted in clinical medicine
• Ultrasound kinetoarteriography (Ware & Laenger, 1965, 1966; McCutcheon & Rushmer, 1967) to detect systolic and diastolic components after cuff occlusion and deflation.	• Require accurate placement over artery underneath the cuff; movement will change position and affect accuracy.
• Penaz method of BP measurement (Penaz J, et al. 1976). Utilizes volume clamp method described by Penaz	• Attempt to measure BP continuously. Resulted in the Finapres®
• Tonometric method of BP measurement (Kemmotsu O, et al., 1991). Flattens the artery to absolve unwanted forces for accurate beat-to-bat BP Measurement	• Requires frequent arm cuff calibration whenever there is change in vessel diameter (since force required to flatten artery changes with change in vessel diameter)
• Vasotrac® Method of BP measurement (Belani KG et al. 1995). Utilizes the principle of maximum energy transfer	• Measures BP over the radial artery by cyclical compression. Frequent BP readings with arterial wave-form display.

Utility Study #2

Large Teaching Hospital in the Southwest

Goal: Assess the impact of BIS on volatile agent requirements and recovery profiles using desflurane and sevoflurane.

Background:

60 patients undergoing laparoscopic surgery received either sevoflurane or desflurane for anesthetic maintenance. A randomly selected BIS treatment group was maintained at a BIS level of 60 $\pm$ 5. The control group followed standard practice without BIS.

Results: BIS-monitored patients showed; >30% reduction in use of volatile agents among BIS titrated group. No difference in analgesic requirements. 30-55% reduction in time required for awakening and extubation among BIS titrated group. (Song D Joshi GP, White PF. Titration of volatile anesthetics using Bispectral Index facilitates recovery after ambulatory anesthesia. *Anesthesiology* October 1997; 87:4.)

Blood pressure (BP) is routinely measured non-invasively during the anesthetic care of patients. Most commonly this is done with an upper arm cuff using either an automatic oscillometric method or by the visual observation of oscillations on an aneroid meter during cuff deflation. The history of BP measurement is quite interesting. Although William Harvey described the circulation in 1628, he did not explain the force that propels blood around the body. But, if one feels a superficial artery, a rough idea of the pressure within it can be gained by feeling the force necessary to compress it. This approximation method has been used by many clinicians even before calibrated arterial-occluding devices became available. Terms such as "thready pulse" or "bounding pulse" reflect the indirect estimation of BP without a device. The non-instrumental palpatory technique of BP measurement has been in vogue for many decades as evidenced by Brunton's report to the British Medical Association in 1909: "The ordinary method of ascertaining blood pressure is to put three fingers upon the radial pulse. The finger nearest the hand prevents the recurrent pulse through the palmar arch, the middle finger feels the pulse and the finger nearest the heart compresses the artery until the middle finger can no longer feel the pulse. The tension is estimated by the greater or lesser amount of pressure required to extinguish the pulse."

Being a subjective phenomenon, it was obvious that there would be significant variations in the interpretation of BP. Thus, Brunton further pointer out that: "Although much information can be gained in this way, the chances of error are considerable, even to an experienced observer and no definite information as to the exact tension (pressure) can be conveyed from one person to another". Therefore, quantification became necessary. The chronological table summarizes the efforts taken by numerous medical and biomedical specialists to try to quantify BP measurement by indirect methods.

During the process of development (see table), it became evident that the application of counterpressure occludes an artery - this was easily identifiable as disappearance of the distal pulse. Secondly, during the application of counterpressure, visible oscillations became evident as the counterpressure was increased; thirdly, with adequate counterpressure blanching could be noticed distal to the counterpressure and release of counterpressure would result in return of colour. As long as these principles were understood, indirect BP could be measured by using one of the following techniques:

- Palpatory
- Flush
- Oscillometric
- Auscultatory

All of the 4 methods above provide reasonably accurate systolic pressure; however, the diastolic pressure is measured with acceptable accuracy by the auscultatory method.

To overcome the practical problems of arm occluding cuffs with air-fluid systems, Riva-Rocci (1896) in Italy and Hill and Barnard (1897) in England introduced air-inflated arm-occluding cuffs. The application of pressure and the appearance and disappearance of a pulse underlying the cuff was easy to measure. The average of pulse-occluding and pulse-reappearing pressures was considered systolic BP. Von Recklinghausen (1901) was instrumental in recommending appropriate cuff sizes for indirect BP measurement by occlusive methods. This is one of the most important factors that influences the accuracy of measurement with the arm cuff technique.

Korotkoff proposed the auscultatory method of blood pressure measurement in 1905 at the time when oscillometric end-points were being verified. The auscultatory method is reasonably accurate over a wide range of blood pressures; however it has been noted to be unreliable during hypotensive states. All of the systems described so far measure BP intermittently and involve occlusion of an extremity, usually the upper arm. When beat-to-beat measurement is required an invasive arterial catheter is placed and a calibrated blood pressure wave-form is displayed on a monitor by means of a transducer amplifier system. Attempts to measure BP continuously in a non-invasive manner resulted in the development of the Finapres and the tonometric methods. Both methods are accurate during steady states. However problems with Finapres accuracy during hypotensive states and the requirement of frequent calibration with the tonometric method have resulted in a lack of appreciation of these 2 systems during the anesthetic care of patients. Recently, a new method to measure BP frequently non-invasively called the Vasotrac has been introduced. The system consists of a wrist module with a pressure transducer. By applying pressure over the radial artery at the distal end of the radius, the pressure transducer assesses the maximum energy transfer of pulsatile beats to determine BP and display a calibtated arterial pressure waveform similar to that obtained directly by a catheter transducer system placed in the radial artery. The system requires 12-15 beats to display the calibrated waveform and arterial BP. The system is as accurate as directly obtained radial artery pressure, has few limitations and may be the answer to frequent non-invasive BP monitoring of anaesthetized patients.

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A Practical Approach to the Unconscious Patient in the Emergency Department

Dr. John K. Gosnold

Hull & East Yorkshire Hospitals, United Kingdom

Introduction

Prior to retiring to bed at night Hans Christian Anderson would always put a little notice on his bed side table..... "I only appear to be dead".

Unconsciousness is not an absolute thing but a progressive process of different levels and difficulty in management.

This paper sets out to clarify the approach to the management of the unconscious patient and to suggest a didactic system of management which will make the process more simple. Communication between professionals is vital for the best treatment of the patient.

It is therefore important to have an objective methodology of measuring the various levels in order to speak the same language and organise effective treatment protocols

Communication:

An old fashioned method of assessing the level of consciousness was to put the patient into 3 Grades:

1. Fully conscious.
2. Unconscious but responds to speech.
3. Unconscious but responds to painful stimuli.
4. Unconscious but with no response.

This method, although acceptable at the time, was imprecise and subject to many variables.

The Glasgow Coma Score was then introduced, allowing for a much more accurate and transferrable system of objective scoring. The GCS was based on the measurement of the different levels of:

1. Eye opening.
2. Response to the spoken word.
3. Response to painful stimuli.

Marks are awarded to allow for a fully conscious score of 15.

Glasgow Coma Score

Eye Opening (E)	**Score**
Spontaneous	4
To speech	3
To pain	2
None	1
Best Motor Response (M)	
Obeys commands	6
Localises to pain	5
Withdrawal to pain	4
Flexes to pain (decorticate)	3
Extends to pain (decerebrate)	2
No response (flaccid)	1
Best Verbal Response (V)	
Orientated	5
Confused conversation	4
Inappropriate words	3
Incomprehensible sounds	2
No sounds	1
Fully Conscious Score	15

ATLS now recognises that even GCS is rather cumbersome for the scoring of unconsciousness in the field and suggests a simplified scoring system of:

Alert

Voice

Pain

Unconscious.

which does seem remarkably similar to the old fashioned method considered to be rather unreliable.

A System

The Traditional approach to the unconscious patient would be to go through a list of diagnoses likely to be the cause for unconsciousness for which various pnemonics exist:

Eg. Stroke
Epilepsy
Morphine and other drugs
injury

Meningitis and other intra-cranial infections
Alcohol
Insulin
Diabetes

Pneumonia and Respiratory failure
Uraemia and Hepatic failure
Psychiatric

Linking to the various systems is another methodology:

Cardiovascular

Arrest
Vasovagal
Arrhythmia
Hypovolaemia

Neurological

Cerebrovascular Accident
Epilepsy
Trauma

Respiratory

Hypoxia
Asphyxia
Carbon monoxide poisoning

Abdominal

Gastro-intestinal bleed
Aneurysm

Endocrine

Diabetes
Others

Toxicology

Overdose
Abuse

Trauma

Head Injury
Hypovolaemia

There are only a limited number of reasons as to why patients present unconscious to an Emergency Room and the final common pathway is the brain.

Dogma

1. Direct local effect - Trauma and Cerebro-vascular accident
2. Insufficient nutrient supply.
4. Insufficient Oxygen supply.
5. Chemicals/Drugs.

Diagnosis

The basis of making a good accurate diagnosis is the framework of:

History

Examination

Investigations.

History

When was the patient last well?

What is the nearest to accurate account of the nature of Illness?

Is the condition improving or getting worse?

Is there any history of Injury?

Has the patient been unconscious or have history of blackouts?

Is there a history of Diabetes?

Is there any possible history of poisoning, either deliberate or accidental?

Is there any history of recent foreign travel?

Is the patient on any drugs, either therapeutic or accidental?

Has the patient any known allergies?

Examination

1. *General*

   The patient should be completely undressed for examination.

   Look for any sign of injury or illness

   Look for injection marks

   Examine the clothes and property thoroughly.

   Look for any warning cards or bracelets.

2. *Skin*

   Colour.

   Temperature and sweating.

   Injections marks.

   Sings of blistering.

   Rashes.

3. *Cardiovascular*

   Bradycardia.

   Tachycardia.

   Atrial fibrillation.

   Hypotension.

4. Respiratory

   Raised respiratory rate.

   Respiratory depression.

   Irregular respiration.

5. Neurological

   Glasgow Coma Score.

   Limb power and tone.

   Pupil size and reflexes.

   Retinal fundi and signs of haemorrhages.

   Scalp, for any sign of injury.

   Neck stiffness.

Investigations

Glucose. Use Bm Stix or Glucometer, but beware variance in raised blood sugar levels.

Blood gases.

Urea and Electrolytes.

Haematological profile

Drug screen.

Radiology. Plain X-ray and CT Scan.

Electrocardiogram.

Treatment

Always stick to the basic principles of:

Airway

Breathing

Circulation

Think constantly of the mechanism and exclude by logic any treatable cause.

Case Examples

1. 16 year old female.
   Eyes open to pain.
   Flexes.

Grunting.
Pupils 8mm.
Divergent squint.
Plantar response extensor.
Fits.
BM Stix - normal
Blood gases - normal
ECG - Supra ventricular Tachycardia

?Diagnosis.

2. 35 year old male.
   Found on floor.
   Smells of Ethanol
   E1, M2, V2.
   Right hemiparesis.
   Pupils normal.
   Occasional fit.
   BM Stix low.

?Diagnosis.

3. 30 year old housewife.
   Collapse whilst ironing.
   No localising signs.
   Frothing at the mouth.

? Diagnosis.

4. 56 year old male.
   Previously healthy - recent diarrhoea.
   Deeply unconscious.
   Shallow respirations.
   Pin point pupils.
   Nil else to find all tests normal

? Diagnosis.

5. 50 year old male
   Collapse at the bus station.
   Respiratory arrest in the Emergency Room.
   Anticoagulant card in pocket.
   Unilateral leg oedema.

?Diagnosis.

Summary

Stick to dogma
Think of the system most likely involved.
Treat any immediately treatable cause.

Status Epilepticus: Emergency Management Protocol

Dr. H.V. Srinivas
Consultant Neurologist, Bangalore Hospital, Bangalore

Status Epilepticus: Definition

A) Two or more seizures without recovery of consciousness between the attacks

or

B) Continuous seizure activity for 30 minutes or more.

Management Protocol

1. ***General*** - Ensure Adequate Airway
   - Maintain BP
   - I.V. 50% 50 ml Glucose *if* Hypoglycemia is suspected.
   - I.V. Thiamine 100 mg *before* Glucose *if* patient is a known alcoholic.
   - Draw Venous Blood for Investigations.

2. ***Pharmacotherapy*** - *(All Drugs Intravenous Only)*

- Start
  - I.V. Diazepam 10 mg (0.2 mg/kg) Adult 10 mg in 4 minutes.

    or
  - I.V. Lorazepam 4 mg (0.1 mg/kg) Adult 4 mg in 4 minutes.
- Followed by
  - I.V. Phenytoin (18 mg/kg) *undiluted* Adult 1000 mg in 20 minutes.
- 5 Minutes Later:
  - If seizures continue (repeat Diazepam / Lorazepam)
- 30 Minutes Later:
  - If seizure continue *"Refractory Status"*
  - Shift to ICU - Ventilatory Support required.
  - I.V. Phenobarbitone (10 mg/kg) Adult 600 mg in 10 minutes.

Administer anyone of the following

a) I.V. Midazolam

Loading 0.1 - 0.3 mg/kg

Maintenance - 2 mg/kg/hour

b) I.V. Pentobarbitone

Loading - 10-20 mg/kg 25mg/minute

Maintenance - 1 to 2 mg/kg/hour.

c) I.V. Thiopentone

Loading - 100 to 250 mg over one minute.

Maintenance - 1000 mg in 500 mg Saline 0.2 mg/kg min

Increase every 5 min till seizures stop or EEG burst suppression.

d) I.V. Propofol

Loading - 1mg/kg

Maintenance - 1 to 3 mg/kg/hour

e) I.V. Lidocaine

Loading 1.5 to 2 mg/kg (100 mg in 2 minute)

Maintenance - 3 to 4 mg/kg/hour

Also treat the underlying cause simultaneously and add symptomatic therapy.

- Antibrain Oedema Measures, Mannitol 20% 100ml x 8th Hrly
- Antibiotics (Pyogenic Meningitis)
- Anticoagulants (Cerebral Venous Thrombosis)
- Correction of Metabolic/Electrolyte Abnormalities.

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(CHC)
11973

Anti Epileptic Drugs in Status Epilepticus

Drug	Loading Dose (mg/kg)	Serum Half Life (in hours)	Onset of Action (in minutes)	Duration of Action (hours)	Maximal Rate of I.V Administration (per minute) Adult dose
Lorazepam	0.05-0.1	20	2-3	4-14	2 mg (4 mg in 4 min)
Diazepam	0.2-0.5	60	1-3	24-72	4 mg (10 mg in 4 min)
Midazolam	0.05-2	1-2	1-5	1-5	0.75 mg (5 mg in 6 min)
Phenytoin	18-20	50	10-30	12-24	50 mg (1000 mg in 20 min)
Phenobarbital	10-20	96	15-60	24-96	50 mg (600 mg in 12 min)

Stroke - Can the Emergency Physician Reduce Deaths and Deficits?

Dr. V. Anantharaman
Head of the Department of Emergency Medicine, Singapore General Hospital, Singapore

Introduction

Strokes are acute cerebrovascular events and a major cause of morbidity and mortality. Strokes leave permanent brain damage and if the victim survives the acute event, he is often left with permanent disability that not only make him a burden to his family, but also to society.

Epidemiology

Stroke is the third leading cause of death in most countries that are strict with their mortality statistics. The incidence of stroke increases exponentially with age and is maximal in the later decades of life. It is probably the leading cause of disability in many societies. Its continuing loss to the economy is enormous and has probably not been clearly elucidated.

Why do strokes occur?

Stroke occurs when one of the following occurs:

a) the blood supply to a portion of the brain/brain stem is interrupted by an ischaemic event (60-80%) such as a thrombus or an embolus,

b) spontaneous haemorrhage (20-40%) occurs in the brain/brain stem. Such haemorrhage may be either intracerebral or subarachnoid.

Many factors contribute to the development of stroke. Some of these are:

a) **Hypertension** is the most major of risk factors and is said to be responsible for upto 70% of strokes. Hypertensives have a three times greater stroke risk than normotensive individuals. It has been estimated that a 7.5 mm Hg increase in diastolic blood pressure is associated with a doubling of the stroke risk. Conversely, a 40% risk reduction can be obtained by lowering diastolic blood pressure by 6 mm Hg.

b) **High serum cholesterol** is said to be related to increased incidence of ischaemic strokes.

c) Just as for coronary heart disease, **cigarette smoking** also has a strong correlation with all forms of stroke.

d) **Obesity** is said to be a contributor to the current relative high incidence of stroke.

e) **Plasma fibrinogen** concentration is a known risk factor.

f) **Diabetes Mellitus.**

g) **Oral contraceptives**, especially preparations with greater than 50 ug of oestrogen.

h) **Elevated levels of homocysteine** are said to predispose to arterial vascular disease and therefore contribute to an increased risk for stroke.

i) **Atrial fibrillation** is a major contributor to cardio-embolic strokes, and the commonest source of these emboli is the atria.

Time course of brain damage in an acute stroke

- An eminently time sensitive disease, stroke results in damage to brain tissue either by direct loss of perfusion, damage by bleeding into the substance of the brain, or by oedema surrounding acutely damaged areas of the brain.
- Within seconds to minutes of the loss of perfusion to a portion of the brain, the generation of the ischaemic cascade results in a central area of irreversible infarction surrounded by an area of potentially reversible ischaemic penumbra.

- At the cellular level, the ischaemic neurons depolarize, ATP becomes depleted and ion transport systems fail. Calcium influx results leading to the release of a variety of neurotransmitters and large quantities of glutamate which further activates N-methyl-D-aspartate and further neurotransmitter release, and the vicious cycle continues. Free radicles are generated and further neuronal damage occurs. Within hours, specific genes are activated, leading to the formation of cytokines and other factors, which in turn cause further inflammation and microcirculatory compromise with extension of the ischaemic penumbra.
- The extreme time sensitive nature of the evolution of stroke means that there are many potential points at which interventions could either halt or retard the progression of the untoward effects of stroke. Such interventions could be in the following areas:
  i. Studies indicate that reperfusion with restoration of cerebral blood flow must occur within 3 hours for the ischaemic penumbra to be saved.
  ii. Neuroprotective agents may block the earliest stages of the ischaemic cascade, and are probably effective only in the very early hours of onset of the stroke.
  iii. Early arrest of the growth of haematoma in the case of haemorrhagic strokes may assist in arresting/slowing further increase of intracranial pressure with its adverse consequence of brain hemiation and resulting neurological deterioration.

Concept of "Brain Attack"

The acute time sensitive nature of brain injury following strokes makes it very similar to the rapid progression of events that occur in cardiac muscle following an acute myocardial infarction. Brain tissue is, perhaps even more fragile than myocardium, and maximizing the salvage of neurons requires a very aggressive approach similar to that which has seen improvements in myocardial salvage over the last decade. It is, therefore, very appropriate to term a stroke as a "Brain Attack" and subject it to conceptually similar time management targets that have become the norm for heart attacks.

The Acute Management of the Stroke Patient

The onset of stroke usually occurs in the home or at the workplace. Because of the time-sensitive nature of stroke, correct management depends on the ability to recognise its symptoms and signs. The management of stroke shall therefore be discussed with a view to appreciating the value of interventions made at every stage of the temporal development and progression of the stroke.

A. Pre-hospital Management

1. There is a need for public education to recognise the very early features of an acute stroke. For ease of response, the following warning symptoms may be used as indicators that a patient may be having a stroke:
   a. Sudden numbness or tingling in an arm or leg
   b. Sudden weakness or paralysis in an arm or leg
   c. Sudden loss of speech
   d. Sudden vision problems
   e. Sudden dizziness
   f. Sudden severe headache
   g. Sudden loss of consciousness (passing out or blacking out for a short time)

   It is important that the public be informed that the warning signs of a brain attack might happen on only one side of the body and that these symptoms indicate that one must get medical help right away by calling for the community's Emergency Ambulance Service.

2. The initial features of a brain attack have to be well taught to ambulance paramedics. Once recognised, the following are the priorities to be adopted by ambulance crew.
   a. Rapid transport to the nearest appropriate Emergency Department is essential to evaluate the patient with initial symptoms.
   b. The following should be initiated by the ambulance crew en-route the hospital:
      i. rapid glucose estimation
      ii. cardiac monitoring
      iii. pulse oximetry
      iv. supplemental oxygen
      v. establish intravenous access
   c. Collect prescription bottles which the patient may have been using and instruct family members or witnesses to go to the ED.
   d. Inform the receiving ED of the impending arrival of the possible stroke patient.

B. Emergency Department Management

1. Assess airway, breathing and circulation. Address any compromise in their status as clinically indicated. Laboured weak respirations especially with hypoxia, would be an indication for intubation and ventilation. High flow oxygen should be provided for all such patients.
2. Patients with possible acute stroke should be "fast-tracked" to allow rapid evaluation. Such evaluation should determine the time when symptoms started. In patients whose symptoms were present upon awakening, their time of symptom onset is estimated from the last time that the patient's neurological status was known to be normal, or the time just prior to going to sleep. Other diseases that may mimic a stroke, such as an epileptic disorder, hypoglycemia, complex migraine, dysrrhythmia or syncope should be excluded.
3. Careful physical examination is crucial in not only determining the probable causes of the stroke, but also documenting as accurately the physical and functional state of the neurological and musculo-skeletal system. Physical examination may also help to distinguish stroke from stroke mimics. The physical examination would consist of the following.
   a. A careful head and neck examination for signs of trauma, infection and meningeal irritation.
   b. A careful search for cardiovascular causes of stroke, including examination of the ocular fundi (retinopathy, emboli and haemorrhage), heart (irregularities, murmurs and gallop), and peripheral vasculature (palpation of carotid, radial and femoral pulses and auscultation for carotid bruits).
   c. The ability to protect the airway should be assessed in patients with a decreased level of consciousness.
   d. A brief, but accurate neurological examination, the goals of which would include:
      i. Confirming the presence of a stroke syndrome
      ii. Distinguishing a stroke from stroke mimics
      iii. Establishing a neurological baseline should the patient's condition improve or deteriorate.
      iv. Essential components of the neurologic examination include evaluation of mental status and the level of consciousness, cranial nerves, motor function, sensory function, cerebellar function, gait and deep tendon reflexes.

v. The skull and spine should also be examined and signs of meningismus should be sought.

vi. Stroke scales, such as the NIH (National Institute of Health) Stroke Scale provide a rapid and reproducible tool for quantifying neurologic deficits in stroke patients and can be useful for following the course of the patient following initial management.

4. Laboratory studies would be required, especially a Full Blood Count (FBC), blood coagulation profile (PT/aPTT), serum electrolytes, and a rapid blood glucose. ECG and a Chest X-Ray should also be obtained. In addition, if the patient is a candidate for thrombolytic therapy, type and cross match should be obtained.
5. All patients with signs of stroke should undergo a noncontrast CT to screen for intracranial bleeding and to rule out expanding lesions such as a subdural haematomas, or other indications for emergent neurosurgery. While conventional MRI may not be as sensitive for intracranial bleeds as CT, newer MRI techniques such as diffusion weighted imaging are being shown to identify ischemic stokes earlier and more reliably than CT scanning.
6. Overaggressive treatment of high blood pressure is not recommended in acute stroke as this may decrease cerebral perfusion pressure and theoretically worsen brain injury especially in the area of the ischemic penumbra and in the setting of increased intracranial pressure.
7. Patients with raised intracranial pressure are candidates for ICP monitoring. Raised intracranial pressure may be treated by either osmotherapy with mannitol, controlled hyperventilation, or with barbiturate coma.
8. Thrombolytic therapy, especially with tissue plasminogen activator (t-PA), if carefully managed, is gradually being shown to possibly result in some benefit if given within three hours of onset of ischemic stroke. Though an increased incidence of intracranial bleeds occurs with such therapy, newer studies that will help to redefine doses at a lower level may help to significantly decrease such adverse consequences. Currently used dose for acute ischemic stroke is 0.9 mg/kg with a maximum dose of 90 mg. Ten percent of the dose is given as a bolus dose and the remainder is given over 60 minutes.
9. Neuroprotective agents have to date not clearly demonstrated benefit in acute stroke patients. Calcium channel blockers, such as nimodipine, free-radical scavengers such as tirilazid and neuronal membrane stabilizers such as citicoline have all been tried. Many newer agents are also being tried in numerous trials currently being conducted. The next five years would be likely to throw more light on the subject of the role of such agents in acute stroke.
10. Anticoagulants and anti-platelet agents are also being demonstrated to have some benefit. However, their role in the overall management of stroke is also currently being defined.
11. For intracranial bleeds and surgically correctible lesions, the earlier institution of such surgery is gradually demonstrating better outcomes.
12. An aggressive protocol for management of Strokes in the Emergency Department can be implemented with the close co-operation of Emergency Medicine, Neurology and Neurosurgery.
13. Early admission of all acute stroke patients to a dedicated "Stroke Unit" has been said to improve outcome.

14. The development of detailed clinical care pathways for the emergency management of acute strokes are gradually pushing the state of art of stroke management and the expectations for outcome closer to what has been achieved for acute myocardial infarctions.

Conclusion

Mortality and morbidity in acute stroke is to a large extent dependent on the efficiency at which the initial emergency management of the stroke patient is carried out. The two principle arenas for such management appear to be the pre-hospital environment, and the Emergency Department. Since Emergency Physicians have a major say in the practice of Emergency care in both these areas, their role in the acute assessment and management of stroke and their potential to positively influence outcomes in acute stroke patients is tremendous.

Approach to Neuroinfections in the Emergency Room

Dr. P. T. Acharya

Professor of Neurology, M.S. Ramaiah Medical College and Hospital, Bangalore

Even after fifty years of discovery of penicillin which once revolutionised the treatment of bacterial infections, infectious diseases continue to remain an important source of morbidity and mortality throughout the world. Infections of the nervous system, in particular, continue to pose diagnostic and therapeutic challenges and frequently present as emergencies. The last two decades have witnessed significant changes in the field of neurodiagnosis, especially, neuroimaging and molecular biology which have enabled us to quickly arrive at the diagnosis and also, the arrival of newer antimicrobial drugs which has changed the treatment perspective. At the same time, newer challenges have surfaced such as the emergence of drug resistant strains of organisms and the growing population of immuno-compromised people especially the HIV infected people and the ones treated aggressively for malignancies and organ transplants. The frequency of opportunistic and mixed infections is steadily growing.

Neuroinfections, in particular, the ones presenting as emergencies carry a high risk of mortality and morbidity. The ultimate outcome and successful treatment of neuroinfections depend on a rapid and accurate diagnosis and an early institution of the necessary treatment. They present with various syndromes[1,2,3,4] such as :-

1. Meningitic syndrome:

This is the most common form consisting of fever, headache, vomiting, nuchal pain, photophobia, signs of meningeal irritation and altered consciousness. Majority of them present in the acute form and occasionally, the chronic form presents acutely because of its complications.

a) Viral[1,2,3,4,5]

- Enteroviruses such as Echo, Coxsackie and Polioviruses.
- Herpes simplex type 2, Lymphocytic choriomeningitis, Varicella, Mumps, Adeno and EB virus.

b) Pyogenic[1,2,3,6,7]

- Neonates-Enteric gram negative bacilli, Group B streptococcus, Listeria Monocytogenes.
- Children (up to the age of 18 yrs.) H. Influenza, Streptococcus Pneumoniae, N. Meningitidis.
- Adults (18-50 yrs. immunocompetent) Streptococcus Pneumoniae, N. Meningitidis, H. Influ.
- Elderly (above 50 yrs. and Immunocompromised)- S.Pneumoniae, Listeria, Gram neg.bacilli
- Post neurosurgical, recent head injury and shunt- Staphylococci, Enteric gram.neg.bacilli, S. Pneumoniae.

c) Bacterial aseptic meningitis - Partially treated pyogenic meningitis, Leptospira, Lyme disease, Mycoplasma

d) Chronic (due to complications) - Tuberculosis, Syphilis, Cryptococci, Toxoplasmosis, CMV, HIV.

2. Encephalitic syndrome:

In addition to fever, headache, vomiting there are early loss of consciousness, seizures and focal deficits. Commonly caused by viruses.

a) Viral[1,2,4,5,8] - (Sporadic)

- H.Simplex type 1, Varicella, CMV, E.B.virus, Mumps, Measles and Rabies.
- (Epidemic)[9] -Arboviruses such as Japanese B., K.F. Disease, Dengue fever.

b) Rickettsial, Lyme disease, Leptospirosis

c) Parasitic - Malaria, Amoebic, and Toxoplasmosis.

3. Meningoencephalitic syndromes:

A combination of features of encephalitic and meningitic illnesses.

4. Space occupation:[1]

Patients present with features of raised intracranial pressure such as headache, vomiting and papilloedema. Focal deficits and seizures may be present. Common causes are Abscess, Granuloma, Subdural Empyema, Epidural abscess and Hydrocephalus.

5. Seizure phenomenon :

Rarely neuroinfections present with seizures which may be in clustures or status epilepticus such as in neurocysticercosis or encephalitis.

6. Vascular syndromes:

Neuroinfections, especially Tuberculous Meningitis, Neurosyphilis and Fungal infections may present with localising or lateralising features due to vascular complications. Other vascular syndromes which present as emergencies are Septic Cavernous Sinus Thrombosis and Rhinocerebral form of Mucor Mycosis.

7. Spinal cord syndromes[1] or Radiculopathy:

Rarely neuroinfections may present as an emergency with paraplegia, quadriplegia, respiratory distress or retention of urine due to spinal cord or root involvement. E.g. Poliomyelitis, Bacterial and Viral myelitis, Spinal Epidural Abscess, T.B. and Enterovirus EV70.[9]

8. Miscellaneous:

Rare emergencies in neuroinfections are sudden visual loss as seen in T.B. Meningitis with optochiasmatic arachnoiditis; a state of shock or bleeding tendency as seen in Dengue fever.

There are various neurological syndromes presenting as emergencies which are the result of neurotoxins such as Tetanus, Botulism and Diphtheria and also the post-infective demyelinative diseases of the CNS. These are not neuroinfections in the true sense and hence connot be discussed here.

In order to arrive at a quick diagnosis one must follow a systematic and scientific approach especially in the case of a neuroinfection presenting as an emergency where time is precious. This should include-

1. Assessment of vital functions and their restoration.
2. Quick bedside evaluation which should include a detailed history, general and neurological examination.
3. Identification of the syndromes mentioned above.
4. Necessary investigations to arrive at the diagnosis.
5. Institution of specific and supportive treatment without delay.

Assessment of Vital Functions and their Restoration:

- Maintain a patent airway
- Ensure adequate oxygenation
- Procure a central venous line
- Ensure adequate hydration
- Maintenance of blood pressure
- Control of seizure
- Antioedema drugs

It is a good practice to send blood samples for CBC, ESR, Glucose, Urea, Creatinine, Electrolytes, LFT,

ABG, Blood culture and peripheral smear for Malarial parasites at this stage itself.

History and Examination

Headache, vomiting and altered consciousness are the common symptoms. Occipitonuchal pain and photophobia favour the diagnosis of a meningitic process whereas early alteration in the conscious level, focal deficits and seizures suggest the possibility of an encephalitic process. History of otitis media, mastoidits, sinusitis, recent head injury, CSF rhinorrhoea and neurosurgical procedures may suggest a septic focus in the near vicinity of the brain resulting in a bacterial infection of the CNS. Evidence of congenital heart disease with right to left shunt, pneumonia, bronchiectasis, lung abscess and bacterial endocarditis suggests a focus of infection which might have resulted in hematogenous transmission of infection to the CNS. History of recurrent meningitis may suggest the presences of a septic focus closes to the brain, CSF leak, Mollaret's meningitis or an immunodeficiency state. Recent gastrointestinal infection, nasal catarrh, exanthema, conjuctivitis or parotitis may suggest a viral etiology. History of having visited an endemic zone may be useful. Dogbite in the recent past with an encephalitic picture and hydrophobia may prompt the diagnosis of Rabies. Knowledge of recent outbreak of an epidemic encephalitis is useful. Presence of high fever with chills and rigors should raise the suspicion of cerebral malaria. History of myalgia, conjunctival suffusion and jaundice in a sewage worker may suggest the possibility of Leptospirosis. Presence of constitutional symptoms may indicate the existence of a chronic illnes such as Tuberculosis or HIV infection. Past history of tuberculosis, diabetes mellitis and exposure to venereal diseases may be useful. Stuffiness of nose, periorbital swelling and blackish nasal discharge with a poorly controlled diabetic state may suggest the possibility of Mucormycosis. Drug history especially of immunosuppressants is useful.

General examination should include recording of pulse rate, blood pressure, respiratory rate and temperature. Severe bradycardia may suggest raised intracranial pressure. Marked pallor with hepatosplenomegaly may suggest malaria. Icterus may suggest the diagnosis of Leptospira, Malaria, viremia or septicemia. Search should be made for the presence of a skin rash which is often seen in viral, rickettsial and rarely in meningococcal infections. Signs of meningeal irritation generally suggests a meningitic illness. Cervical matted lymph node enlargement may raise the possibility of a tuberculous illness.

Fundal examination for presence of papilloedema should be done. A detailed neurological evaluation should be undertaken which should include,

1. Assessment of level of consciousness on the Glasgow Coma Scale. This is useful in prognostication as a deeper level of coma signifies a grave prognosis.
2. Identification of any lateralising or localising signs which may help in planning the investigation.
3. Identification of any cranial nerve palsy which may help in the diagnosis.
4. Recognition of any seizure activity.

Some neuroinfections have certain characteristic deficits which are useful in making a diagnosis. Tuberculous meningitis has frequent involvement of ocular cranial nerves. Herpes simplex encephalitis often has prominent behavioural changes and lateralising signs. Cryptococcal meningitis often presents with very severe headache. Seizures are very common with cerebral malaria, viral meningitis, neurocysticercosis, tuberculoma and cerebral abscess. At the end of neurological examination generally one can categorise a given case into one of the above mentioned syndromes.

Investigations:

The choice investigative modalities should be very precise in order to arrive at an early diagnosis. One must quickly go through the results of the tests that are already asked for:

Certain hematological tests are useful under special circumstances such as:-

1. Reported blood smears for malarial parasites whenever an encephalitic picture is associated with anemia and liver function derangements.
2. Antibody titres against Dengue infection should be done whenever an encephalitic illness is associated with neutropenia or thrombocytopenia.
3. Antibody (IgM) titres against leptospiral infection may be asked for whenever a neuroinfection is associated with jaundice, myalgia and conjunctival suffusion.
4. Weil-Felix titre if rickettsial infection is suspected.
5. Paul Bunnel test if Infectious Mononeucleosus is considered.
6. HIV titres if there is a clinical suspicion of AIDS.
7. VDRL, TPA or FTA tests.

An X-ray of chest is often a very useful test which may reveal the presence of tuberculousis in the lungs or pleura, miliary tuberculosis, lung abscess or empyema.

Neuroimaging study such as C.T. scan or MRI scan of the brain is an essential and very useful test in intracranial infections. These studies are mandatory in the presence of focal deficits. Even while pyogenic meningitis is strongly suspected it is desirable to have a C.T. scan done in order to identify associated abnormalities such as cerebral abscess, ventriculits, subdural effusion or empyema. C.T. scan of the brain may appear normal or may show features of diffuse cerebral edema in viral and pyogenic meningitis. Herpes Simplex encephalitis is often associated with temporal or frontal lobe focal edema, necrosis or hemorrhage. MRI is superior to C.T. scans in this case. Presence of enhancing basal exudates, hydrocephalus and infarcts favour the possibility of T.B. meningitis. Brain imaging is useful in identifying granulomatous disorders and neurocysticercosis.

Cerebrospinal fluid examination is of great importance in the diagnosis of neuroinfections. Pressure is often elevated. CSF is clear and colourless in viral infections and aseptic meningitis. Turbid CSF is suggestive of pyogenic meningitis. An opalescent CSF with a cobweb is in favour of T.B. meningitis. CSF may be rarely hemorrhagic as in the case of hemorrhagic encephalitis. Gram stain, Z.N. stain and India Ink preparations are very useful in identifying the microorganisms. CSF should be sent for culture and sensitivity studies in case of pyogenic and tuberculous infections. Antibody studies are done in case of tuberculosis, neurocysticercosis, and viral encephalitis (Herpes Simplex, Japanese B Viruses). Antigen detection is undertaken in case of T.B. meningitis, and Herpes Simplex infections. Polymerase chain reaction (PCR)[2] studies are extremely useful in the rapid diagnosis of Herpes Simplex, and tuberculous infections.

Pyogenic meningitis has certain characteristic CSF abnormalities such as cell count usually over 1000/ cmm with predominant polymorphs, mild to moderate elevation in the protein level and the glucose level usually less than 30% of the blood level. CSF glucose level below 20 mg% is characteristic. However, partially treated pyogenic meningitis may not have all these features but a very low CSF glucose level, elevated CRP and lactate level[6] will favour the diagnosis. Tuberculous meningitis is often associated with lymphocytic pleocytosis, moderate elevation in the

protein level and a mild reduction in the glucose level. Reduction in the chloride level is not reliable. Cryptococcal meningitis has more or less similar features. CSF glucose level may be significantly low and India Ink preparation is positive. Most of the viral neuro infections show an aseptic meningitis picture with lymphocytic pleocytosis, mild or no elevation in the protein level and normal glucose level.

In an HIV infected patient one must look for opportunistic and mixed infections such as CMV, Toxoplasma and Atypical Mycobacteria. Mycobacterial CSF culture sensitivity study is also recommended as one is anticipating drug resistant strains. Whenever Rabies is suspected a corneal smear or cervical skin biopsy may be useful. Electroencephalographic studies are useful in the diagnosis of Herpes Simplex encephalitis. Characteristic features are spike and slow wave discharges and occasional periodic lateralised epileptiform discharges arising from one temporal lobe.

Whenever a spinal cord syndrome is suspected an MRI study is necessary to exclude a compressive clause. A CSF analysis may be necessary if an infective condition is suspected. Estimation of VDRL titre in the CSF may be done. Polio antibody titres may be estimated if there is a motor flaccid paralysis.

Treatment

The overall outcome in neuroinfections depends largely on the institution of effective treatment at the earliest. The treatment should include the administration of specific antimicrobial drugs and also the supportive therapy. In some cases neurosurgical intervention may be also necessary in addition to the medical treatment. A mention of emergency treatment has already been mentioned earlier in order to stabilise the vital functions:

a) Supportive treatment

1. If the patient is unconscious Mannitol infusion is given as an antiedema treatment.
2. Adequate control of seizures with appropriate antiepileptic drugs.
3. If there is any evidence of metabolic acidosis as seen on ABG studies an adequate amount of parenteral sodium bicarbonate is administered.
4. Occasionally hypoglycemia may be noticed which may require immediate correction.
5. If the patient is febrile antipyretics and tepid sponging may be necessary.
6. Adequate hydration is necessary.
7. If there is any bleeding tendency fresh blood transfusions or platelet concentrates are given.

b) Specific treatment

1. Pyogenic meningitis

If the clinical picture is suggestive of acute meningitis and if the CSF is turbid high dose antibiotics with adequate penetration into the CSF space are started empirically.[1,2,3,6]

Neonates - Cefotaxime plus ampicillin

Infants and children - 3 rd-generation cephalosporin

Adults

Healthy, immunocompetent, community-acquired

- 3rd-generation cephalosporin or pencillin G or ampicillin

Nosocomial (postneurosurgical or head injury)

- 3rd-generation cephalosporin plus oxacillin or vancomycin plus aminoglycoside.

Shunt related meningitis - 3rd-generation cephalosporin plus vancomycin plus aminoglycoside

Immuno compromised - 3rd-generation cephalosporin plus ampicillin.

Once the Gram's stain or the culture sensitivity is available the antibiotic may be changed depending upon the microorganism identified.[1,2,3,6]

Meningeal pathogen	Antibiotic	Alternatives
N. meningitidis	Penicillin G or ampicillin	3rd gen. cephalosporin
S. pneumoniae	Penicillin G or 3rd gen. cephalosporin	Ampicillin Vancomycin
H.influenzae	3rd gen. cephalosporin	Ampicillin plus chloramphenicol
Grop B Streptococci	Penicillin G or 3rd gen. cephalosporin	Ampicillin
Enterobacteriaceae	3rd gen. cephalosporin	Ampicillin plus aminoglycoside
Pseudomonas	Ceftazidime plus aminoglycoside	Piperacillin plus Aminoglycoside
S. aureus (methicillin sensitive)	Oxacillin	Nafcillin or vancomycin or fosfomycin
S. aureus (methicillin resistant)	Vancomycin	
Coagulase negative staphylococci	Vancomycin	
L.monocytogenes	Ampicillin (plus aminoglycoside)	Trimethoprim-sulphamethoxazole

Whenever aminoglycosides are recommended it may be worthwhile considering once-daily therapy in order to minimise the vestibular and renal toxicity.[6] In children with pyogenic meningitis dexamethasone is administered intravenously at 0.15 mg/kg every 6 h for 4 days in order to minimise the meningeal inflammation following bacterial lysis due to antibiotics. The first dose of dexamethasone should be given before the first dose of the antibiotic.[1,6]

2. Viral meningoencephalitis

If clinically viral encephalitis or meningitis is suspected the treatment is largely symptomatic. However, in Herpes simplex and zoster infections parenteral Acyclovir is extremely useful. If clinically Herpes simplex type 1 infection is suspected and there are imaging and EEG characteristic features IV Acyclovir can be empirically started. If PCR, antigen or rising antibodies are present a full course of therapy for 14 days is given.

3. Tuberculous meningitis

If the onset of the illness is subacute or chronic and if the CSF picture is suggestive of TBM a course of anti TB treatment with four drugs and steroids are started.

4. Cryptococcal meningitis

If India Ink preparation is positive in the CSF a course of Amphotericin B with flucytosine is given.

5. Cerebral Malaria[10]

In a case of acute encephalitis with fever, rigors, anemia, icterus and splenomegaly empirically one can start the patient on IV Quinine, Artesunate (Falcigo) or Mefloquine.

6. Opportunistic infections

In immunocompromised patients often there exist mixed infections such as Mycobacterium tuberculosis, atypical Mycobacteria, Crytococcus, Toxoplasma and Cytomegalo virus. Mycobacterial culture sensitivity may be useful in treating drug resistant atypical Mycobacteria. IV Gancyclovir is useful in CMC infection. In CNS Toxoplasmosis[10] a course of pyrimethamine, folinic acid and sulphadiazine is recommended.

7. Neurocysticercosis

A course of Albendazole or Praziquantel with steroids is recommended.

8. Miscellaneous

If Leptospirosis is suspected penicillin G and tetracycline are recommended. If rickettsial infection is identified a course of tetracycline is useful. In case of cavernous sinus thrombosis a course of high dose of antibiotics to cover against staphylococci are used. Fungal infections of the nervous system are treated with appropriate antifungal agents. In cases of HIV infections antiretroviral drugs are also used. If neurosyphilis is confirmed procaine penicillin or aqueous penicillin is recommended. In case of cerebral abscess 3rd generation cephalosporin, aminoglycoside and metronidazole are administered.

d) Neurosurgical intervention

Cerebral abscess, subdural empyema, epidural abscess, large cerebral space occupying granuloma, Rhinocerebral Mucormycosis and hydrocephalus need surgical intervention in addition to appropriate antimicrobial treatment.

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Emergency Management of Acute Renal Failure in Adult Patients - Current Perspectives

Dr. Lloyd Vincent
Associate Professor and Head, Department of Nephrology
St. John's Medical College, Bangalore

Introduction

Acute renal failure (ARF), is diagnosed when there is a decline in renal excretory function and increase in plasma urea and creatinine concentrations occur over hours or days. Chronic renal failure is a slow progressive irreversible destruction of the nephron mass leading ultimately to end stage renal disease needing dialysis and or transplantation. Symptoms in either case would depend on the rapidity of development, the etiology of renal failure and the extent of renal damage.

In the emergency setting the diagnosis of renal failure is usually made when the patient is referred as renal failure or has a history, or signs and symptoms of renal failure. However often the diagnosis cannot be made reliably on clinical grounds alone and an elevated plasma concentration of urea or creatinine is found for the first time in the emergency department.

Guidelines for A Clinical Approach to Handling Renal Failure in an Emergency Setting.

Given the common and serious nature of the problem, a practical clinical approach to the patient with renal failure, is something which all who have medical responsibility for acutely ill patients should be familiar.

The questions to be asked are:

Is the renal failure acute or chronic?

What are the clinical and biochemical features of renal failure?

What is the renal size?

Is prerenal failure and acute tubular necrosis or haemodynamically-mediated acute renal failure the cause?

- Is urinary obstruction a possibility?
- Are glomerulonephritis, interstitial nephritis, vasculitis, drug induced renal disease or other rarities possible?
- Is an acute renal vascular event possible?
- Are there any life-threatening complications, in particular hyperkalaemia and or pulmonary oedema?
- Has the required urgent medical treatment already been administered?
- Is there a need for renal replacement therapy?
- What are the indications for renal replacement Therapy?
- What are the goals of renal replacement therapy?
- What are the available choices of renal replacement therapy?
- Is there a need to refer to an appropriate specialist unit for associated problems ?
- Prognosis - will the patient survive?
- What are the important preventive aspects of in hospital acute renal failure?

Is the Renal Failure Acute or Chronic?

Some clinical features can help in deciding whether renal failure is acute or chronic: a history of vague ill health of some months duration, of nocturia, of pruritus; the findings of skin pigmentation, anaemia, hypocalcaemia and hyperphosphataemia, evidence of long-standing hypertension or neuropathy, would all suggest chronicity. Radiological evidence of renal osteodystrophy is only found in patients with obviously

long-standing chronic renal failure. Elevated urea leads to increased carbamylation of haemoglobin in circulating red cells, low levels indicate that the biochemical derangement is of recent origin and that renal failure is acute. In cases where there is uncertainty a diligent search for previous notes and biochemical information may prevent the waste of a great deal of time, effort, and money in unnecessary investigations.

The fact that a patient had a 'bit of protein in the urine', biochemical evidence of renal failure in the past or sonologically bilaterally contracted kidneys noted at a prior medical examination would almost certainly indicate a long-standing renal disease suggestive of chronic renal failure. The only basis for excluding the possibility of pre-existing chronic renal impairment with absolute confidence is the knowledge of a previous normal measurement of renal function. In the absence of the above the renal failure could be acute or still a previously undetected chronic renal failure.

If acute renal failure is present, then the clinical presentation is almost invariably dominated by that of the condition which precipitates it and a high index of clinical suspicion is required to diagnose acute renal failure at an early stage of its development. On this background, all patients admitted to hospital with an acute illness should be considered to be at risk of having undiagnosed acute renal failure, and whilst they remain acutely ill to be at substantial risk of developing acute renal failure. The condition is frequently accompanied by oliguria [urinary volume < 400 ml/day], sometimes by anuria [< 100 ml/day], but is not infrequently associated with normal urinary volume. History of urinary volume, colour or intake may not be forthcoming. Symptoms and signs attributable to the accumulation of urea and other toxins within the body are non-specific, and they are not evident until renal failure is far advanced. Inability to excrete salt and water can lead to the development of peripheral and/or pulmonary oedema. Failure of renal acid excretion can lead to the development of systemic acidosis, and induce a very characteristic Kussmaul respiration, if diabetic ketoacidosis has been excluded. In cases where the cause of acute renal failure is not obvious, the importance of excluding urinary obstruction and intrinsic renal disease (nephritides and vasculitides) at an early stage is emphasized, since delay in making these diagnoses and initiating appropriate treatment can lead to the tragedy of avoidable permanent loss of renal function. Those who have some pre-existing chronic impairment of renal function are particularly susceptible to acute exacerbations. This group includes all elderly patients, in whom a combination of low muscle mass and low dietary meat consumption may conspire to maintain an apparently 'normal' plasma creatinine, despite a reduction in glomerular filtration rate to as little as 25% of that expected in a healthy young adult.

In a large number of cases, however, there are no features of the history or examination that enable the physician to make a confident declaration that the renal failure is acute or chronic when a patient is first clinically assessed. Given the lack of reliable clinical indicators, all patients admitted to hospital with an acute illness should have their plasma concentration of urea or creatinine measured to establish whether or not they have impaired renal function.

What are the Clinical And Biochemical Features of Renal Failure?

Any patient with suspected renal failure would need certain basic investigations to confirm the diagnosis and determine the etiology of renal failure.

A hemogram would reveal anaemia that is usually present and haemoglobin typically is around 8 to 9g/dl. This is caused by impaired erythropoiesis, attributable both to erythropoietin deficiency and to 'toxic' effects of uraemia on the marrow, and to reduced survival of red blood cells or bleeding. The leucocyte

count is often elevated but not always, indicating the presence of infection. Eosinophilia is rarely seen, but when present should lead to consideration of the diagnosis of acute interstitial nephritis. The platelet count is often slightly reduced ($100–150 \times 10^9$/l) especially if DIC is present, and defective platelet function contributes towards a tendency to bleed. A number of distinctive haematological pictures need to be recognized in those with acute renal failure especially with Disseminated intravascular coagulation, Hemolytic Uremic Syndrome and myeloma because they have diagnostic and therapeutic significance.

The clinical diagnosis of ARF renal failure, acute or chronic, is made when plasma concentrations of urea and creatinine are elevated. The clinical picture is almost certain to be dominated by the primary underlying etiology of renal disease. In the early stages there are few symptoms attributable to renal dysfunction however unsuspected, but potentially fatal, hyperkalaemia is the greatest danger. Particularly rapid increases are to be expected when there is extensive tissue damage or hypercatabolism, as in rhabdomyolysis, burns, and sepsis.

Fifty percent of patients may be nonoliguric. In the later stages important biochemical changes include the development of uraemia with anorexia, nausea and vomiting (occasionally diarrhoea), muscular cramps, metabolic acidosis, hypocalcaemia, hyperphosphataemia, and signs of encephalopathy including a 'metabolic' flapping tremor (asterixis), depressed consciousness, and *grand mal* convulsions. Skin bruising and gastrointestinal bleeding may occur worsening hyperkalemia. Acidosis is sometimes the metabolic abnormality most obviously necessitating urgent institution of renal replacement therapy. Uraemic haemorrhagic pericarditis occurs much less frequently in acute than in chronic renal failure, but when it does it is another potentially fatal complication. In acute renal failure the renal systems for excreting acid fail, leading to the development of metabolic acidosis. This is usually modest in degree (plasma pH 7.2–7.35), but can on occasion be severe, manifesting as sighing Kussmaul respiration and/or with circulatory compromise. (see below).

Many patients with acute renal failure have hypocalcaemia and hyperphosphataemia, probably secondary to disordered vitamin D metabolism. In acute renal failure the plasma sodium is usually normal, since deficits of sodium are usually matched by those of water: hence extracellular fluid volume may be reduced, but plasma sodium concentration remains unchanged. On occasion, however, intake of water perhaps driven by hypovolaemia may exceed the rate at which it can be excreted (which is depressed when the glomerular filtration rate is very low) and hyponatraemia results. The retention of uric acid, sulphate, and magnesium occurs in acute renal failure, but these biochemical abnormalities are rarely clinically significant. Concentrations of uric acid can, however, be particularly high in rhabdomyolysis and tumour lysis, when they may warrant specific treatment.

What is the Renal Size?

In any patient presenting with renal failure of unknown cause, one of the first investigations should be imaging to determine renal size, and also to confirm that two kidneys are present. In most instances, chronic renal disease causes the kidneys to shrink, and it is the finding of 'two small kidneys' that most often indicates the presence of previously unsuspected chronic renal disease.

On a **plain radiographic film** of the abdomen the outline of the kidneys can often be traced, the normal length being 3.7 ± 0.4 times the height of the second lumbar vertebral body. Better definition of the renal outline could be obtained by **intravenous urography**, but this procedure is not recommended

in patients with acute renal failure because high doses of contrast medium are necessary to obtain images in those with poor renal function, the images obtained in such circumstances tend to be of relatively poor quality, and there is concern that the contrast media used can be nephrotoxic. The standard imaging technique used to assess renal size is **ultrasound** which is non-invasive, has no known deleterious effects, and is also the first-line imaging technique to assess whether a patient has urinary obstruction. The normal kidney length measured by this method is 10 to 12 cm. **Radioisotopic methods** ([^{131}I]iodohippurate, [^{99}Tc^{m}]DTPA, [^{99}Tc^{m}]DMSA) have been used to determine renal size in patients with renal failure, but they have not been found to be clinically helpful and are not routinely employed in this context. **Computed tomography** can also be used to measure the size of the kidneys, but is rarely required for this purpose alone.

Is Prerenal Failure and Acute Tubular Necrosis or Haemodynamically Mediated Acute Renal Failure the Cause?

The vast majority of cases of acute renal failure will fall into the categories of prerenal failure or acute tubular necrosis/haemodynamically mediated acute renal failure.

The term **prerenal failure** is used when renal dysfunction is entirely attributable to hypoperfusion and restoration of renal perfusion leads to rapid recovery. Circumstances predisposing to prerenal failure are almost invariably associated with increased plasma levels of antidiuretic hormone acting on the collecting duct to increase tubular reabsorption of both water and urea , hence plasma urea increases out of proportion to creatinine in prerenal failure. Plasma urea may also appear to be disproportionately increased with sepsis, steroids, tetracycline (catabolic effect) and gastrointestinal haemorrhage (protein meal).

The term **acute tubular necrosis/ haemodynamically mediated acute renal failure** is used, to describe a particular clinical scenario: **(a)** it is seen in specific clinical contexts, frequently involving circulatory compromise and/or nephrotoxins; **(b)** urinary abnormalities usually suggest tubular dysfunction; and **(c)** essentially complete recovery of renal function is expected within days or weeks in most cases (if the patient survives the precipitating event). Acute renal failure of this type can be seen after virtually any episode of severe hypovolumia, cardiac dysfunction or circulatory compromise due to sepsis or septic shock causing hypotension, but not all causes of circulatory derangement are equally devastating to renal function. Primary impairment of cardiac performance, for example following myocardial infarction, may cause plasma creatinine to increase somewhat, but rarely causes renal failure of sufficient severity to require renal replacement therapy. By contrast, haemodynamic upset caused by sepsis frequently does. Many patients with haemodynamically mediated acute renal failure do not have a single cause for their renal failure: the kidney most typically fails when subjected to multiple insults. Dehydration, hypotension, sepsis or septic shock, excessive exposure to aminoglycosides, and pigmenturia are significant acute insults. An additive interaction between insults is seen, and the severity of acute renal failure is related to the number and magnitude of acute insults.

Prerenal failure and acute tubular necrosis/ haemodynamically mediated acute renal failure almost invariably occur in the context of circulatory disturbance, in patients who are shocked. **If the patient is not, or has not recently been, in such a state, then it is not likely that their acute renal failure is attributable to either of these conditions and it becomes particularly important to exclude obstructive or intrinsic renal causes.** If the circulation

is, or has been, deranged, then prerenal failure or acute tubular necrosis or haemodynamically mediated acute renal failure are likely.

The urinary indices try and discern whether a patient is in prerenal failure or acute tubular necrosis/ haemodynamically mediated acute renal failure based on analysis of the urine. The measurements most frequently made are of sodium, urea, and creatinine concentrations in plasma and urine, together with urinary and plasma osmolality. The values obtained are used to derive a number of variables: **the ratios of urine to plasma concentrations of urea, creatinine and osmolality; fractional sodium excretion** (FE_{Na} = [urinary sodium × plasma creatinine × 100]/[plasma sodium × urinary creatinine]); and **'renal failure index'** ([urinary sodium × plasma creatinine]/urinary creatinine. In the 'typical' case of **prerenal failure** the urinary electrolytes are said to reflect the response of normal tubules to impaired renal perfusion. There is avid retention of sodium and water, leading to low urinary sodium and high urinary urea and creatinine concentrations, together with a high urinary osmolarity. Restoration of renal perfusion leads to rapid improvement in renal function. By contrast, in the 'typical' case of **acute tubular necrosis/ haemodynamically mediated acute renal failure,** the urinary sodium concentration is elevated and the urinary urea and creatinine concentrations and urinary osmolality are relatively low features suggestive of tubular dysfunction. In such instances, whatever the treatment, renal function rarely improves rapidly. Not all, however, have found urinary tests to be so discriminating and able to separate those with a clinical diagnosis of prerenal failure from those with acute tubular necrosis on the basis of urinary measures. If is now agreed that **urinary indices have little merit in routine clinical practice and do not reliably predict outcome in acute renal failure,** and treatment is begun on exactly the same lines whether the diagnosis is of prerenal failure or of acute tubular necrosis. The response to initial treatment retrospectively defines the diagnosis and determines further management.

Is Urinary Obstruction a Possibility?

One of the merits of the traditional division of the causes of acute renal failure into prerenal, renal, and postrenal types is that it demands consideration of the possibility of urinary obstruction, a common cause of both acute and chronic renal impairment. It is extremely important that **obstruction should not be missed because it is readily treatable and, if neglected, causes permanent renal damage.** Even if there are no positive features in the history or examination to suggest obstruction, the diagnosis should be seriously considered in all cases where another positive diagnosis cannot be made. The presence of anuria or of alternating polyuria and oligoanuria are helpful clues. Despite the production of an apparently satisfactory urinary volume, renal function may still be significantly impaired due to obstruction.

A thorough urological history and examination is mandatory in those with acute renal failure in whom the cause is not obvious. Apart from asking the patient the obvious questions about whether they have ever had haematuria, passed urinary stones, had renal colic, had prostatic symptoms, had retroperitoneal or pelvic surgery etc., it is worthwhile probing these aspects of the history in more detail. In a patient with prior undetected unilateral renal obstruction (pelvic surgery, papillary necrosis,stones or malignancy) with permanent loss of function of that kidney, an obstruction of the single remaining functional kidney would develop acute renal failure as a consequence of its obstruction. **Palpation and percussion to see whether the bladder is distended** are often neglected and proper attention to this part of the physical examination should be given, also to

examination of the genitalia (enlarged prostate or gynecologic malignancies) and rectum.

Renal imaging by ultrasonographic examination of the kidneys and bladder is the method of choice in most centres to investigate for the presence of obstruction. However ultrasound detects dilatation of the renal calyces and pelvis, not obstruction, and the diagnosis can be missed either because the calyces fail to dilate or do so minimally. If, despite a 'negative' ultrasound report, the clinician remains concerned that obstruction is a possible cause of acute renal failure, a number of options should be considered. Recognizing that obstruction without dilatation is possible, and also that the quality of image obtained by renal ultrasonography is highly variable depending on the patient, the equipment, and on the operator in the first instance the case should be discussed with the person who made the original ultrasound examination and the scan repeated to see if appearances have changed. If doubt still persists, then further investigations to be considered include **computed tomography, cystoscopy with retrograde ureteropyelography, and (rarely) antegrade nephrostomy. Radioisotope studies, such as DTPA renography with frusemide** injection, can be useful in the diagnosis of urinary obstruction in those with normal or only slightly impaired renal function. In some cases, imaging alone may fail to resolve the problem unequivocally, and in these very rare instances it will be necessary to instigate a 'trial of urinary drainage'. This requires the renal pelvis(es) to be drained by either antegrade nephrostomy(ies) or retrograde stent(s) to see if this leads (over a few weeks) to functional improvement. Substantial recovery of renal function can occur after the relief of long-standing obstruction, up to 4-5weeks of anuria.

Needless to say, if obstruction is diagnosed then it should be relieved by **bladder catheterization, percutaneous (antegrade) nephrostomy or cystoscopic retrograde insertion of ureteric stents,** as a prelude to definitive treatment (where possible) of the underlying obstructive lesion. If acute renal failure is caused by bilateral renal obstruction, then the obstruction to both kidneys should be relieved. This may require bilateral antegrade nephrostomies. The most common causes of urinary obstruction causing acute renal failure are renal calculi, retroperitoneal fibrosis, malignant diseases of the uterine cervix, prostate, bladder and rectum, benign prostatic hypertrophy, and conditions that cause massive retroperitoneal and pelvic lymphadenopathy (lymphoproliferative conditions and secondary carcinoma).

One question that often arises is **'how rapidly renal obstruction needs to be relieved'**, the simple answer is **'as soon as possible'.**

Are Glomerulonephritis, Interstitial Nephritis, Vasculitis, Drug Induced Renal Disease or Other Rarities Possible?

In cases where the cause of acute renal failure is not obvious, the importance of excluding intrinsic renal disease (nephritides and vasculitides) at an early stage is emphasized, since delay in making these diagnoses and initiating appropriate treatment can lead to the tragedy of avoidable permanent loss of renal function

The clinical history and examination may indicate that a patient is likely to have glomerulonephritis or vasculitis as a cause of acute renal failure particular attention should be paid to a history or signs of a skin rash; to arthralgias or arthritis, to myalgias or muscle tenderness and swelling; and to eye, ear or nose problems. These aspects of the history and examination can be of great significance in indicating that a patient has some form of multisystem disorder, vasculitic process, or rhabdomyolysis. Obtaining a thorough drug history is also of crucial importance: penicillins

and non-steroidal anti-inflammatory agents, which in many countries are available without prescription, are the most common causes of acute interstitial nephritis A urine microscopy would show a renal inflammatory process, which although rare has critically important management implications, in any patient with unexplained acute renal failure which may be due to acute glomerulonephritis (of various types), renal vasculitis (of various types), accelerated-phase hypertension or (rarely) interstitial nephritis.. Ten to fifteen ml of urine should be spun at 1500 rev/min for 5min, all but 1 ml of the supernatant carefully discarded, and the pellet resuspended. Examination, looking primarily for red-cell casts should be made under high power, preferably after counterstaining and if present, a renal biopsy, together with other specialized blood tests (antiglomerular basement-membrane antibodies, antineutrophil cytoplasmic antibodies, antinuclear factor antibodies etc.) will be required to establish a precise diagnosis. **It is important to try to ensure that the simple facilities required for urine microscopy are readily available to all who care for acutely ill patients.**

Is an Acute Renal Vascular Event Possible?

Elderly patients with acute renal failure includes many who have widespread atheromatous vascular disease. Some of these patients will be dependent upon a single functioning kidney, the other having been lost as a result of occlusion of the renal artery at some time past. Imaging will demonstrate one kidney of normal size and one which is small, less than 8 or 9cm in length sonologically. Although acute renal failure may be due to acute tubular necrosis in these cases, there is clearly the possibility that it could be precipitated by acute occlusion, thrombotic or embolic, of the renal artery supplying the solitary functioning kidney. This is most commonly seen in hypertensive individuals. Risk factors that have been invoked include exposure to angiotensin-converting enzyme inhibitors, reduction in blood pressure secondary to antihypertensive agents or volume depletion, and radiological procedures requiring exposure to contrast media and (sometimes) the instrumentation of the aorta. The diagnosis of acute renal vascular occlusion is supported if the patient complains of flank pain, has had macroscopic haematuria, if there is complete anuria, or if the patient does pass some urine by the finding of a urinary sodium concentration equal to that in plasma. The best technique to demonstrate acute renal-artery occlusion remains arteriography. Revascularization of a kidney that has a developed collateral blood supply can restore function and get a patient off dialysis many weeks after acute occlusion of the renal artery, fibrinolytic therapy can also be effective; but in those without developed collaterals the kidney cannot be expected to recover from this insult unless intervention is possible within a matter of a couple of hours.

Are There Any Life-threatening Complications, in Particular Hyperkalaemia and or Pulmonary Oedema?

Hyperkalaemia

Unsuspected hyperkalaemia is the greatest danger, since this rarely produces any recognized symptoms whatever before inducing cardiac arrest. Hyperkalaemia is most commonly dangerous in the context of acute renal failure and is important because it can cause cardiac arrest without warning. Patients may occasionally suffer from muscular weakness or paralysis, but these symptoms are rarely prominent, and if they do occur their significance is rarely appreciated (except in patients on long-term dialysis who have had them before).

As the plasma potassium concentration increases this leads to a reduction in the transmembrane

potassium concentration gradient and a reduction in the resting membrane potential. It also acts to shorten the duration of the cardiac action potential by increasing the velocity of cellular repolarization, possibly by altering membrane permeability to potassium, however not all patients are equally susceptible to the cardiac effects of hyperkalaemia

Patients with severe hyperkalaemic cardiotoxicity should be given intravenous calcium: initially 10ml of 10per cent calcium gluconate over 60s, repeated until the electrocardiogram shows clear signs of improvement. From a clinical viewpoint, it is important to recognize that the effect of calcium infusion is relatively transient. After giving calcium to those with life-threatening hyperkalaemia, and in cases of moderately severe hyperkalaemia, the plasma potassium concentration can be reduced by 1 to 2 meq/l over 30 to 60 min by giving an intravenous infusion of glucose and insulin (50ml of 50per cent glucose with 10 units of rapidly acting insulin over 15min). The insulin acts by stimulating the Na–K-ATPase pump in skeletal and cardiac muscle, liver, and (possibly) in fat, thus driving potassium into cells. The purpose of the glucose is to prevent symptomatic hypoglycaemia.

Intravenous sodium bicarbonate (50–100 ml of 4.2 per cent solution over 15min) as an adjunct or alternative to glucose and insulin treatment is often used. Its effect, typically a reduction of plasma potassium concentration of 1 to 2 meq/l, has traditionally been ascribed to an increase in blood pH. However, the routine use of intravenous sodium bicarbonate to treat hyperkalaemia is not recommended due to the following reasons. Firstly, many patients with acute renal failure are volume overloaded, and the treatment necessitates the administration of a considerable sodium load. Secondly, hypertonic solutions of sodium bicarbonate are extremely irritant, and their extravasation can cause extensive chemical burns: they must only be given into central veins. Thirdly, in some patients the treatment can induce a dramatic reduction in the plasma ionized calcium, with the risk that this might precipitate epileptic seizures. Fourthly, the treatment has no advantages over glucose and insulin. The one circumstance in which intravenous hypertonic sodium bicarbonate would seem to be appropriate in the treatment of hyperkalaemia is when this is accompanied by hypotension and profound acidosis, such that it seems very likely that acidosis is contributing to the perilous circulatory state.

Stimulation of B_2-adrenergic receptors leads to activation of the Na–K-ATPase pump and the movement of potassium into cells. This property has been exploited to treat hyperkalaemia in patients with renal failure. Both intravenous salbutamol (0.5 mg over 15 min) and nebulized salbutamol (10 or 20 mg) can reduce plasma potassium concentration by a mean of about 1meq/l over 30 to 60 minutes. It must be emphasized that none of the treatments described above removes potassium from the body. They may be sufficient as treatment for hyperkalaemia on their own if rapid recovery of renal function can be anticipated, for example following resuscitation of those with prerenal failure or relief of urinary obstruction. However, if rapid recovery cannot be expected, then hyperkalaemia will recur over a period of 2 to 4 hours as potassium leaks back out of cells, with further doses of calcium, glucose, and insulin tending to have diminished efficacy. Here the period of temporary respite from dangerous hyperkalaemic cardiotoxicity must be used to instigate treatment that will remove potassium from the body. In less severe cases, cation-exchange resins may suffice (sodium or calcium polystyrene sulphonate, 15 gm in water by mouth or 30gm in methylcellulose as a retention enema every 6hours, in both cases with precautions against the treatment inducing constipation), but in most some form of dialysis or haemofiltration will be required.

Pulmonary Oedema

The most serious complication of salt and water overload in uremia is the development of pulmonary oedema. Severe cases are dramatic. The patient is terrified, restless, and confused. Examination reveals cyanosis, tachypnoea, tachycardia, widespread wheeze or crepitations in the chest, and a gallop rhythm (if the heart can be heard at all). Investigation demonstrates arterial hypoxaemia and widespread interstitial shadowing on the chest radiograph. The immediate management is to sit the patient up, which redistributes fluid from the pulmonary to systemic circulations, and give oxygen by face mask in as high a concentration as possible. Frusemide, or other diuretic agents, are of little benefit in producing diuresis in cases of pulmonary oedema associated with renal failure, although they have some vasodilator action that can be beneficial. Intravenous morphine in small doses (2.5 mg, repeated depending upon the response) is very helpful as both a vasodilator and an anxiolytic. Definitive treatment requires the removal of excess fluid pulmonary oedema being one of the indications for emergency haemodialysis or haemofiltration.

Has the Required Urgent Medical Treatment Already Been Administered?

Many patients with acute renal failure are extremely ill, and the **first priority in management is to treat any problems be they renal or otherwise that are life threatening.** Renal physicians must remember not to concentrate on the kidney to the exclusion of all else: if the patient is not breathing properly, then the first priority is to remedy this, if necessary by **intubation and ventilation.** The two complications of renal failure that come into the life-threatening category are **hyperkalaemia and pulmonary oedema which requires urgent attention.** The next priority is the prompt diagnosis and treatment of ***hypovolaemia and other electrolyte imbalances.*** If features suggestive of **intravascular volume depletion** are present, then the deficit should be corrected rapidly. Attempts to cannulate the central veins should not be made in those who are obviously hypovolaemic. The fluid infused should be of a type that remains substantially within the intravascular compartment (blood, colloid, saline); it should mimic as closely as possible the nature of the fluid lost; and it should be given as rapidly as the tubing and vascular access will permit. The patient should be observed continuously and the infusion stopped when features of volume depletion have disappeared.

Loop diuretics, of which frusemide is the most commonly used, act primarily to inhibit function of the sodium/potassium/chloride cotransporter in the luminal membrane of the thick ascending limb of the loop of Henle

1. The countercurrent flow of blood in the vasa rectae (important for the mechanism of urinary concentration) creates a state in which the oxygen tension in the deeper parts of the cortex and the medulla of the normal kidney is very low. The medullary thick ascending limb may therefore be operating on the verge of anoxia, and particularly susceptible to ischaemic/hypoxic damage if the circulation is perturbed. By inhibiting sodium transport, loop diuretics reduce the metabolic work done by this segment of the renal tubule, and might thereby protect it against ischaemic/hypoxic damage.
2. By increasing the flow of urine down the tubules, loop diuretics may flush out intratubular casts and reduce tubular obstruction.
3. Tubuloglomerular feedback is the process whereby an increased delivery of chloride to the macula densa of a nephron leads to a reduction in the filtration rate of that nephron. Loop diuretics inhibit this process, and may therefore preserve filtration rate in cases of acute renal failure.

4. Renal blood flow is reduced in most cases of haemodynamically mediated acute renal failure. In normal individuals loop diuretics reduce renal vascular resistance and increase renal blood flow, in part via effects on renal prostaglandin production. If they have the same actions in those with, or at risk of, acute renal failure (which is not proven), then they may be beneficial.

Intravenous mannitol is a potent osmotic diuretic, inducing a large natriuresis and diuresis in those with normal renal function. As with loop diuretics, there are a number of theoretical reasons why its effects may be beneficial in cases of acute renal failure.

1. An increase in flow rate through the tubules might flush out intratubular casts.
2. Mannitol may increase renal blood flow.
3. Damage to renal cells causes them to swell, causing vascular congestion and impairing blood flow. The osmotic effect of hypertonic mannitol may prevent this.
4. The same action that reduces cell swelling causes expansion of the intravascular volume and reduces the haematocrit, which may confer protection against ischaemic insult.
5. Mannitol may reduce the influx of calcium into mitochondria that occurs after ischaemia, and thereby preserve mitochondrial function in this situation.
6. Toxic free-radical species are generated during reperfusion of tissue after ischaemia. Mannitol can act as a free-radical scavenger.

Dopamine is an important modulator of renal function. In normal individuals an intravenous infusion of dopamine at a low dose (1–5 μg/kg per min) causes an increase in renal blood flow, glomerular filtration rate, and sodium excretion. It should be noted, however, that whilst such an infusion is frequently referred to as 'renal dose' dopamine, this is a misnomer, implying action on the kidney and nowhere else, whereas some of the effects on renal function are undoubtedly attributable to inotropic action on the heart. There are no randomized controlled trials of its use in patients with established acute renal failure

A wide variety of other agents has been shown to ameliorate the development of acute renal failure, usually when given before, or at the same time as, the causative insult. The following treatments fall into this category: calcium-channel blockers; natriuretic peptides; ATP-MgCl2; glycine and alanine; antioxidant defence strategies; manoeuvres designed to modulate the nitric oxide and endothelin vascular control systems; manoeuvres designed to modulate the action of lipid-derived mediators; and growth factors.

In patients with established renal failure, as a working rule, **fluid intake** in those with acute renal failure should be limited to the volume of the previous day's urine output, plus the volume of other measurable fluid losses, plus 500 ml for insensible losses (an allocation that may need to be substantially increased in the presence of fever or in hot environments). However, fluid-balance charts are frequently inaccurate: thus unthinking adherence to the 'output plus 500 ml' rule can lead to problems. There is no substitute for careful, twice-daily clinical examination for signs of intravascular volume depletion or excess, supplemented by accurate daily weighing to gauge overall net fluid balance, with an intelligent, flexible response to the findings.

Sodium requirements in patients with acute renal failure are very variable. Those who are oliguric may lose, and therefore require, only 15 to 30meq/day. Those who are polyuric may excrete very considerable amounts of sodium (many hundred meg/day), and run the danger of developing intravascular volume depletion if these are not replenished (with the risk of thereby exacerbating their acute renal failure). In the polyuric phase of acute tubular necrosis/haemodynamically mediated acute renal failure the kidneys do not have the capacity to modulate sodium

excretion in the normal manner, such that the urinary sodium concentration is typically in the range 50 to 70meq/l. Hence, if urine output is 3 litres/day, over 200 meq of sodium may be required to prevent negative balance. On occasion the urine output in polyuric acute renal failure can be extremely high (over 5 litre/day is not uncommon). These cases can be difficult to manage: if the therapeutic response is to administer an even greater quantity of fluid (output plus insensible losses), then it is possible to contrive a vicious circle whereby ever-increasing urinary output s met by ever-increasing fluid infusion. Such a danger should be recognized in the polyuric patient, and a luid-replacement strategy employed that avoids this scenario, perhaps by limiting input to a volume equal o the urinary output alone, and allowing other fluid osses to establish a mild overall negative balance. Regular clinical examination and weighing should alert he physician to the risk of development of significant ntravascular volume depletion.

Because hyperkalaemia is one of the great angers in those with acute renal failure, it is essential o check plasma potassium concentrations at least aily; in those with hypercatabolism or gastrointestinal leeding, or who require surgery, more frequent stimations are advisable. In oliguric cases, high-otassium foods should be avoided. By contrast, in olyuric acute renal failure, very substantial urinary sses of potassium can occur (many hundred meq/ ay), and it can be difficult to replace these in a manner at prevents the plasma potassium concentration vinging over a wide range. In these circumstances, easurement of the urinary potassium concentration n be helpful in estimating how much is required, t a patient passing 0.5 litre of urine per hour or ore may need it to be infused at a rate of 40 to meq/h, necessitating central venous access, very equent measurement of the plasma potassium ncentration, and extremely close medical pervision.

Emergency renal replacement therapy is to be considered at once if indicated. Finally, in acute renal failure **the specific diagnosis and treatment of the underlying condition** should be addressed: if this persists untreated then renal function will not improve. In patients with **chronic renal failure** once stable, renal replacement therapy is individualized in the form of **dialysis or transplantation.**

Is there a Need for Renal Replacement Therapy?

Any patient with acute or chronic renal failure would require renal replacement therapy if indicated and cannot be managed conservatively. In **unstable patients with chronic renal failure or acute renal failure** the conditions, constraints and objectives of treatment are notably different from the **stable chronic renal failure situation** in that the former group of patients are usually hospitalized, access to blood is often obtained via a temperory catheter inserted into deep vessels, hemodynamic instability is common and further hypotensive episodes must be prevented to avoid additive acute ischemic injury in the acute situation. Hence clearly specific and personalized renal replacement strategies must be developed to meet the individual needs of the unstable patient.

What Are The Indications For Renal Replacement Therapy?

Indications can be clinical and biochemical and are not considered as individual values to determine need for dialysis but guidelines aiding a total clinical picture to the concerned nephrologist.

Clinical:

- Encephalopathy
- Pericarditis
- Pulmonary odema
- Uremic bleeding
- Anuria > 48 hours (relative)

Biochemical:

- Creatinine > 8 mg %
- Urea > 200 mg %
- Bicarbonate < 10 mg %
- PH < 7.2
- Serum Potassium > 7 mg%

What are the Goals of Renal Replacement Therapy?

The goals of RRT in uremia consist of

- Restoration of sodium and water balance.
- Control of azotemia.
- Restoration of acid-base balance.
- Satisfactory control of serum potassium, calcium and phosphorus levels.
- Adequate nutritional support: caloric intake 30-35kcal/kg/24hours, protein intake 1.2g/kg/24hours or more if needed.
- Correction of anemia.
- Prevention of bed rest complications.
- Relative facilitation of renal function recovery by using biocompatible dialysis membranes.

What are the Available Choices of Renal Replacement Therapy?

Two broad categories of RRT used for the treatment of uremia in the emergency namely **The Continuous Low Flow and Intermittent High flow methods** In both cases several options (Table-1) are available. The choice of therapy in each individual situation is usually guided by the hemodynamic status of the patient, severity of renal failure, co-morbidity and on the advantages and drawbacks of the concerned procedure (Table-2).

Table 1: Various RRT modalities and their principal mechanism(s) for solute exchange,

Modalities/phenomenon	Continuous	Intermittent
Diffusion	Haemodialysis CAVHD/CVVHD	Haemodialysis IHD
Convection	Isolated ultrafiltration SCUF Haemofiltration (post/predilutional HF) CAVH/CVVH	Isolated ultrafiltration IUF Haemofiltration (post/predilutional HF) IVVH
Diffusion + convection	Haemodiafiltration (post/predilutional HDF) CAVHDF/CAVHDF	Haemodiafiltration (post/predilutional HDF) CAVHDF/CAVHDF

IDH, intermittent haemodialysis; IUF, isolated ultrafiltration; HF, intermittent haemofiltration; HDF, intermittent haemodiafitration; SCUF, slow continuous ultrafiltration; CAVH/CVVH, continuous arteriovenous or venovenous haemofiltration; CAVHD/CVVHD, continuous arteriovenous or venovenous haemodialysis; CAVHDF/CVVHDF, continuous arterivovenous or venovenous haemodiafiltration.

Table 2: Advantages and drawbacks of continuous and intermittent RRT

Advantages	Drawbacks
Continuous RRT	
• More physiolgical process • Important cumulative clearances • Better haemodynamic tolerance • Flexible fluid removal capacity • Inflammatory mediator removal (?)	• Lower efficiency per unit of time • Prolonged immobilization • Relatively higher risk for bleeding • Relatively higher risk of circuit clotting • Nursing workload • Arterial catheter hazards (arteriovenous modalities) • Induced hypothermia (?), hypophosphotaemia
Intermittent RRT	
• Available in most centres worldwide • Higher efficiency per unit of time • Easier mobilization • Flexibility of the dialysis machine	• More limited haemodynamic tolerance • Relative restriction for fluid removal • Less physiological process • Fluctuations of the internal milieu

The continuous low flow methods are performed over prolonged periods one or more days, these have a lower instantaneous efficacy which is balanced by its continuous mode of operation. They can be performed in a spontaneous **arteriovenous circulation** (CAVHD, CAVH and CAVHDF) or **venovenous circulation** (CVVHD,CVVH and CVVHDF). (Figure-1). Continuous RRT is performed using highly permeable synthetic membranes, at relatively low blood flows and hence can be performed in patients with hemodynamic instability. The vascular access is usually a percutanous central access and the choice of anticoagulation depends on the associated risk factors and the clinical picture.

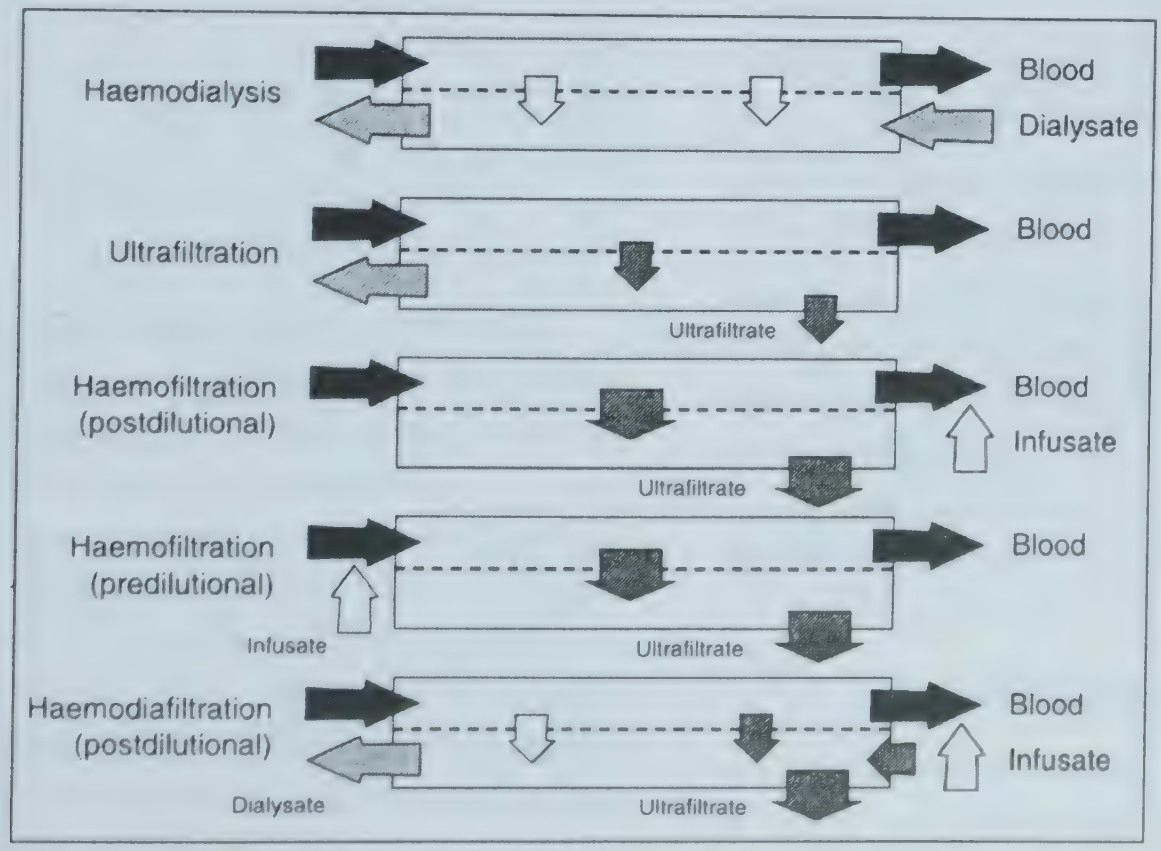

Figure 1: Schematic representation of various RRT modalities used for ARF.

Continuous RRT is particularly for the following situations:

- Congestive cardiac failure or other fluid overload states resistant to conventional medical managemant
- Severe electrolyte disorders
- Certain drug intoxications like lithium.
- Lactic acidosis can be corrected gently with continuous RRT using bicarbonate solutions.
- Acute respiratory distress syndrome may be improved by continuous RRT by depleting the lungs of excess water.
- Extensive burns with acute renal failure.
- Multiorgan failure syndrome with acute renal failure.
- Sepsis for cytokine removal or adsorption.

The intermittent high flow methods (HD, HF and HDF) are performed over short periods of 4-6 hours daily or every other day and have a high solute clearance efficacy in view of the relatively higher blood flow rates maintained. The vascular access is obtained using temporary venovenous double lumen or double venous catheters. The synthetic membranes used have lesser permeability and large surface area. Heparin is the standard anticoagulation used unless contraindications exist.

Is There A Need To Refer To An Appropriate Specialist Unit For Associated Problems?

Often management of these patients in the emergency would require a multidisciplinary team approach depending on the etiology of the renal failure or the presence of comorbid conditions. It may be a surgical intervention in the case of an acute abdomen or a neurosurgical intervention in the case of a trauma. The management would often differ or need to be modifyied as per the specialist opinion or as the situation warrants.

Prognosis-Will The Patient Survive?

Acute renal failure is a serious condition: the mortality of all cases that required renal replacement therapy being about 40 per cent. However, it is clear that not all patients are at equivalent risk, and for many reasons it is important to know whether a particular patient or group of patients falls into a higher or lower category of risk. This information has many implications: most crucially for the counselling of individual patients and their relatives.

The most important prognostic factor in those with acute renal failure is whether or not the kidneys are the only organ system that has failed. Of the other organs, the lungs are the ones that most commonly fail in conjunction with the kidneys, and in a large number of studies of patients requiring dialysis for acute renal failure it has been shown that the need for artificial ventilation has a massive negative effect on outcome.

What are the Important Preventive Aspects of Hospital Aquired Acute Renal Failure?

One of the aims of basic emergency care is to try and **prevent the development of acute renal failure** which **should begin in the emergency**, or, failing that, to recognize its development at an early stage. This requires the careful monitoring of **intravascular volume, fluid input and output, daily weighing (where practicable), measurement of lying and standing (or sitting) blood pressure (at least once daily), and regular estimation of the plasma concentrations of creatinine, urea, and electrolytes.**Care must be taken when potentially **nephrotoxic drugs** are administered. In conjunction with regular repeated clinical examination of the patient, the records must be scrutinized and the test results appraised every day.

Elective surgical procedures should not be performed on those who are acutely ill, but there are occasions when surgery is essential to such patients without it they will not survive. This scenario is accompanied by a very high risk of acute renal failure, the chances of which can be minimized by scrupulous attention to the avoidance of hypovolaemia and unnecessary exposure to nephrotoxins. There is no substantial evidence that giving frusemide, mannitol, dopamine or any other agent confers additional protection.

Renal Emergencies in Pediatrics

Dr. Kishore D. Phadke

Pediatric Nephrologist, Dept. of Pediatrics, St. John's Medical College Hospital, Bangalore

The renal emergencies in Pediatrics are discussed with regard to the fluid balance disturbances, electrolyte & acid-base disturbances, hypertensive crisis, renal replacement therapies, and the role of mannitol, diuretics, and dopamine.

Fluid Balance Disturbances

The patient's state of fluid balance (hypovolumic, euvolumic or hypervolumic) should be assessed. Weight, blood pressure, heart rate, skin turgor and capillary refill are each used to assess the intravascular volume. Urinary indices like fractional excretion of sodium may be helpful. If the patient is intravascularly volume depleted, 20 ml/kg of normal saline or Ringer's lactate can be infused rapidly as a fluid challenge which may be repeated. If there is no urine output after the fluid challenge, furosemide may be used. If the response is poor, this may be the time to consider putting a central line, use of inotropes etc.

If the child has fluid overload, fluid restriction and fluid removal with dialysis or hemofiltration may be instituted if the child does not respond to diuretics. The circulating blood volume should be maintained taking into consideration ongoing fluid losses, urine output and insensible fluid losses. During recovery phase of acute renal failure, substantial fluid and electrolyte losses may occur requiring replacement.

Electrolyte & Acid-base Disturbances

a) *Hyponatremia:* - Symptomatic hyponatremia needs correction. This can be done using the formula - weight in kg. x 0.6 x (130 - serum sodium) - over 48 hours using hypertonic saline. Initially hypertonic saline may be given rapidly at the rate of 1-2 ml/kg/hour till serum sodium reaches 120 meq/lit. Dilutional hyponatremia additionally needs fluid restriction and removal.

b) *Hypernatremia:*- It is usually associated with excessive free water losses or polyuric states with limited access to free water or due to excessive sodium intake. This needs to be corrected gradually over 48 hours using the appropriate formula.

c) *Hypokalemia:*- This could be a serious problem if there are ECG changes (ST, T wave changes, U waves) or if there is respiratory paralysis or paralytic ileus or in patients with digoxin toxicity or hepatic encephalopathy. For these circumstances, hypokalemia should be rapidly corrected, the rate of administration not to exceed 0.5 meq/kg/hour.

d) *Hyperkalemia:*- This can be life-threatening due to disturbances in cardiac rhythm (tall peaked T waves, increased PR interval, widened QRS complexes, ventricular fibrillation).

Measures to combat hyperkalemia:-

1) Stop all intake of potassium - oral and parenteral.
2) Calcium gluconate 10% 0.5-1.0 ml/kg over 5-10 minutes with cardiac monitoring.
3) Sodabicarbonate - 1-2 meq/kg diluted.
4) Glucose 0.5 gm/kg with insulin 0.1 unit/kg over 30 minutes.
5) Nebulisations with β_2 agonists.
6) Kayexalate - 1 gm/kg PO or PR in sorbitol.
7) Dialysis

e) *Metabolic acidosis:-* If it is decompensated and/or if the child is obtunded with compromise of respiratory compensation, sodabicarbonate administration is indicated. However if there is hypernatremia or fluid overload or renal failure, early dialysis for correction of acidosis is advisable.

Hypertensive Crisis

Aim of management is to reduce blood pressure not more than 25% in first 1-2 hours. Measures that may be taken to reduce blood pressure include:-

a) Sublingual nifedipine: 0.25-0.5 mg/kg sublingually.

b) Sodium nitroprusside: intravenously 0.5-1.0 µg/kg/min with hemodynamic & thiocyanate level monitoring.

c) Labetalol: 0.25 mg/kg intravenously.

d) Diazoxide: 2-5 mg/kg iv.

Renal Replacement Therapy

Nephrologist needs to be consulted if renal replacement therapy is contemplated. The options are:

1) Peritoneal dialysis

2) Hemodialysis

3) Hemofiltration:- Especially useful in hemodynamically unstable patients - CAVH, CAVHD, CVVHD, CVVH.

This modality of treatment is useful for rapid fluid removal. When dialysis also done simultaneously, the procedure is called hemodiafiltration.

Role of Mannitol, Diuretics, Dopamine

1) Mannitol

0.5-1.0 gm/kg may increase intratubular urine flow to limit tubular obstruction. It also acts as a scavenger of reactive oxygen molecules. However, lack of response to mannitol may result in hyperosmolality and congestive cardiac failure may be precipitated.

2) Furesemide

1-5 mg/kg increases urine flow rate to decrease intratubular obstruction. It however inhibits Na-K-ATPase which limits oxygen consumption in already damaged tubules with low oxygen supply.

3) Low dose dopamine

0.5-5.0 µg/kg min improves renal blood flow by promoting vasodilatation. However studies have not demonstrated that such therapy improves survival or reduces the need for dialysis in patients with acute renal failure.

Care of the Patient with Fulminant Hepatic Failure

Gerard S. Kamath
Department of Anesthesiology, Mayo Clinic, Rochester, Minnesota 55905

Definition and Terminology

Fulminant hepatic failure is one of the most catastrophic conditions in medicine. Patients with the disease can become extremely ill very rapidly and require all the sophisticated support systems available in today's intensive care unit (ICU). Fulminant hepatic failure is characterized by severe liver dysfunction and hepatic encephalopathy without preexisting symptomatic liver disease. In contrast, the patient with chronic symptomatic liver disease suffers a progressive declining course that may be punctuated by episodes of exacerbation of liver dysfunction or encephalopathy. A correctable cause of the exacerbation (e.g., spontaneous bacterial peritonitis or gastrointestinal bleeding) is frequently identified and dealt with, or an alteration of medical therapy may provide comfort and control of the patient's symptomology.

Many different nomenclatures have been used to describe fulminant hepatic failure, O'Grady's frequently used terminology (1) is based on a large series of patients who were seen over a period of 13 years at King's College Hospital in London (Table 1). This scheme divides patients into three groups: hyperacute, acute, and subacute. Encephalopathy remains as a necessary fearure in the diagnosis of fulminant hepatic failure in O'Grady's scheme. Hyperacute liver failure is defined as that in which hepatic encephalopathy occurs within 7 days of the onset of jaundice. Hyperacute liver failure is associated with a high incidence of cerebral edema and, with medical management, carried a relatively high (36%) survival rate in the King's College series. Acute liver failure is diagnosed when the interval between jaundice and encephalopathy is 8 to 28 days. In the King's College series, acute liver failure was associated with a high incidence of cerebral edema but with a much poorer survival rate (7%). Subacute liver failure occurs when the jaundice-to encephalopathy interval is greater than 4 weeks but less than 6 months. Subacute liver failure is characterized as having a relatively low incidence of cerebral edema and a midrange survival rate of 14%.

Although survival rates for patients with fulminant hepatic failure have improved since the availability of orthotopic liver transplantation, fulminant hepatic failure remains a very critical condition that can take a previously healthy individual to terminal illness in a matter of days.

Etiology

The causes of fulminant hepatic failure include infections, drug and toxin exposure, and other miscellaneous etiologies. Infectious causes include primarily viral hepatitides (A, B, C, D super infection, non-A, non-B, non-C (2, 3), Epstein-Barr virus (4), herpes simplex virus (5), cytomegalovirus (6), and adenovirus (7)). Drugs are responsible for approximately 15% of cases of fulminant hepatic failure, with acetaminophen overdose being the most frequent cause. Isoniazid (8), tricyclic antidepressants (9), volatile anesthetics, and nonsteroidal antiinflammatory drugs (10) have also been implicated. Toxins are responsible for less than 2% of cases of fulminant hepatic failure and include *Amanita phalloides* (11), carbon tetrachloride (12) and aflatoxin (13).

Miscellaneous causes of fulminant hepatic failure include ischemic liver-cell necrosis due to shock (14); obstruction of the hepatic veins, as can occur in the

Table 1. O'Grady's nomenclature of fulminant hepatic failure

Group	Time from onset of jaundice to encephalopathy in days	Frequency of cerbral edema (%)	Frequency of ascites	Survival with medical therapy in percent
Hyperacute	<7	Frequent (69)	Rare	36
Acute	8-28	Frequent (56)	Rare	7
Subacute	34-84	Rare (14)	Frequent	14

Budd-Chiari syndrome (15) and veno-occlusive disease of the liver (16); Wilson's disease (17); hyperthermia (18); autoimmune hepatitis; partial hepatectomy (19); microvascular steatosis, which can occur from acute fatty liver of pregnancy (20); and Reye's syndrome (21). In infants, in-born errors of metabolism (22), such as galactosemia, hereditary fructose intolerance, and tyrosinemia, must also be considered in the etiology of fulminant hepatic failure.

Clinical Presentation

Because patients with fulminant hepatic failure characteristically are well until the relatively recent onset of illness, they typically do not have the stigmata that are usually associated with chronic liver disease. External signs of malnutrition are seldom present. Spider angiomata are characteristically absent, and ascites, although sometimes present, is usually not a striking feature of the physical examination. What characterizes the patient with fulminant hepatic failure is the presence of hepatic encephalopathy. Although hepatic encephalopathy does occur in patients with chronic liver disease, encephalopathy does not have the importance or prognostic implications that it has in patients with fulminant hepatic failure.

The rapidly failing liver has a significant detrimental effect on all other organ systems. The reason for this far-reaching effect on other organ systems is quite probably the systemic circulation of soluble mediators (e.g., such as cytokines) or secondary products of cytokine-activated cells (e.g. nitric oxide) (23). This adverse condition may be the result of the failing liver's inability to clear cytokines or, possibly the result of a release of activated inflammatory cells from the declining liver (24). In addition, as the liver's function declines, it is increasingly unable to perform host-defense functions involving the opsonic capabilities of such proteins as fibronectin, C-reactive protein, and α_1 antitrypsin, or to clear gut-derived translocated bacteria and endotoxin (25).

Neurologic Complications

The presence of hepatic encephalopathy is essential to the diagnosis of fulminant hepatic failure and is traditionally subdivided into four stages (Table 2). Grade 1 is the most benign, but progression to Grades 2, 3 and Grade 4 indicates a deteriorating neurologic condition.

Table 2. Clinical grading of hepatic encephalopathy

Grade of encephalopathy	Neurologic findings
1	Behavioural changes without changes in level of consciousness
2	Gross disorientation, slowing of mentation, occasional decrease in level of consciousness
3	Suppressed level of consciousness but responsive to painful stimuli, no meaningful communication
4	Unresponsive to pain, decerebrate posturing

The etiology of hepatic encephalopathy, both in fulminant hepatic failure and chronic liver disease, is thought to be a result of the presence of false neurotransmitters (phenylalanine, tyrosine, and other amines) (26). Aromatic amines are normally largely catabolized by monoamine oxidase in the liver and cleared from portal blood, but when hepatic function is impaired, these amines can gain entrance into the nervous system and replace normal neurotransmitters.

Hepatic encephalopathy can present significant problems for the patient with liver dysfunction. Alterations in mental status can result in the patient having difficulties with mobilization of secretion and airway maintenance, which may require endotracheal intubation. However, in-patients with fulminant hepatic failure, neurologic complications can continue beyond those found with hepatic encephalopathy alone.

Cerebral edema accounts for 38% to 80% of the fulminant-hepatic-failure-associated mortalities in autopsy studies (27-29). Cerebral edema has been documented in 48% to 86% of patients with fulminant hepatic failure when intracranial (ICP) monitoring is used (30, 31). By anticipating and detecting the cerebral edema and increased ICP, the clinician can affect the overall outcome for these patients.

Therapies directed at the treatment of intracranial hypertension include head elevation, hyperventilation, diuretic therapy, and intravenously administered barbiturate infusions (see section on treatment) (32).

ICP monitoring may be required for the proper titration of these therapies. When Lidofsky et al. (30) integrated ICP monitoring into their care of patients with fulminant hepatic failure, they achieved an overall survival rate of 56.6% (92% in those receiving liver transplants). This rate is compared to a 26% survival rate found in a survey of numerous other centres (33). Despite this indirect evidence of improved survival with the use of ICP monitoring, a prospective randomized investigation showing the value of ICP monitoring has not yet been carried out. ICP monitoring also provides a useful piece of data to avoid transplanting patients with intractable intracranial hypertension and low cerebral-perfusion pressure. ICP monitoring has been reported to be relatively safe. In an analysis combining the experience of 68 centres, 262 patients with fulminant hepatic failure and ICP monitoring had an incidence of intracranial hemorrhage of 3.1% with epidural monitors and 18% with subdural bolts (34). The incidences of fatal hemorrhage were 1.3% and 5%, respectively. Infectious complications were minimal, and malfunction that required repositioning occurred in 7.4% of patients.

The use of barbiturates has been shown to be effective in treating refractory intracranial hypertension in patients with fulminant hepatic failure (35), especially when renal failure is present. The effects of barbiturates result from their anaesthetic properties, which reduce the oxygen requirements of the brain, and from their ability to decrease cerebral blood flow, which leads to a decrease in the volume of the intracranial contents and, hence, a fall in ICP. The use of computed tomography (CT) scans for the detection of cerebral edema in these patients is controversial. Many have felt that cerebral edema, as noted on CT scan of the head, occurs too late in the course of the disease to be of much diagnostic assistance; however, Wijdicks et al. recently looked retrospectively at the CT scans of 12 patients with fulminant hepatic failure (36). Without knowledge of the patients' clinical data and using a brain-edema-severity score, they found evidence of cerebral edema on the initial scans in 7 of 8 patients with Grade 3 and 4 hepatic encephalopathy. No cerebral edema was identified in the patients who presented with Grade 1 or 2 hepatic encephalopathy.

Cardiovascular Complications

Similar to patients with chronic liver disease, patients with fulminant hepatic failure also have the

hyperdynamic circulatory state. This condition of elevated cardiac output (CO) and depressed systemic vascular resistance (SVR) was first described in 1953 by Kowalski and Abelman (37). The etiology of this hemodynamic condition is unknown; however, it has been suggested that this condition may result from a supply-dependent oxygen delivery, similar to that which occurs in septic shock (38). An additional or alternative cause of the hyperdynamic circulatory state could be the failing liver's inability to remove vasoactive mediators. To state that the hyperdynamic circulatory state is a "complication" may be a misnomer, as the elevated CO appears to be facilitative in supplying blood and oxygen to the failing vital organs (most notably the liver). In fact, if the hyperdynamic circulatory state is absent, the clinician should begin investigating the cause of the patient's inability to generate a high CO. Complications of the hyperdynamic circulatory state may occur in those individuals who are elderly or who have underlying cardiac lesions and cannot respond to the demands of a hyperdynamic circulation and, thus, develop cardiac failure.

Pulmonary Complications

Patients with chronic liver disease may experience a hypoxemic condition resulting from pulmonary-vasculature dilation, called the hepatopulmonary syndrome (39). The incidence of the hepatopulmonary syndrome in patients with fulminant hepatic failure is unknown; however, an isolated case has been reported (40). When hypoxemia does occur in the patient with fulminant hepatic failure, it usually is the result of pulmonary infiltrates from fluid overload (often the result of the transfusion of multiple coagulation products) and pneumonia. When pulmonary infiltrates occur, they need to be treated in a vigorous manner (e.g., bronchoscopy with broncho alveolar lavage), since these patients are immunocompromised and infections with opportunistic organisms can occur. The hyperdynamic circulatory state can result in mild pulmonary hypertension, which can be diagnosed on the basis of an elevated CO. However, severe pulmonary hypertension, as can occur in patients with chronic liver disease, has not been described.

Many patients with fulminant hepatic failure do eventually require endotracheal intubation and mechanical ventilation. These interventions are usually made for airway-management purposes and for the application of hyperventilation in the patient with increased ICP.

Complications of Coagulation

The liver is the primary source of the procoagulant factors, except factor VIII. Production may be impaired in both fulminant hepatic failure and chronic liver disease. In addition, the patient with hepatic disease is frequently vitamin-K deficient, due to malabsorption of fat-soluble vitamins, impaired enterohepatic circulation, malnutrition, or abnormal bowel flora, so vitamin-K-dependent factors II, VII, IX and X may be independently depressed. The liver is also compromised in its abilities to remove circulating factors that are important in the coagulation cascade (e.g., activated clotting factors, fibrinolysins, etc.).

Platelet counts are usually depressed in patients with liver disease. Thrombocytopenia in the patient with chronic liver disease most typically results from portal hypertension and hypersplenism. This particular etiology of thrombocytopenia is rare in the patient with fulminant hepatic failure, and much of the thrombocytopenia seen in these patients results from an ongoing disseminated intravascular coagulopathy (DIC) and fibrinolysis. When disorders of platelet function occur in fulminant hepatic failure, they are usually associated with the occurrence of renal insufficiency.

The treatment of this multifactorial coagulopathy in fulminant hepatic failure is sometimes extremely

difficult. Some assistance in disease management and coagulation-product transfusion can be obtained from the results of coagulation monitors that provide the clinician with an overall assessment of this multifactorial process. The thromboelastogram is such a device and has been used over the past 2 decades in the care of patients undergoing liver transplantation (41). Its use can be easily transferred to the ICU (42).

Infectious Complications

Patients with fulminant hepatic failure are significantly immunocompromised and, hence, are at risk for the development of opportunistic infections. A fatal complication occurring with a considerable frequency is disseminated Aspergillus infection (32). The loss of the liver's ability to clear gut-derived translocated bacteria and endotoxin can result in the circulation of these and other inflammatory mediators, which can initiate the systemic inflammatory response syndrome (SIRS) (25). Selective bowel decontamination sterilizes the patient's bowel of potentially pathogenic organisms (aerobic bacteria and fungi), while preserving a potentially protective anaerobic flora. Selective bowel decontamination has been used in liver-transplant recipients and in other ICU patients. The results of bowel decontamination in these varied patient groups have been mixed (43).

GI Complications

GI hemorrhage frequently occurs in patients with fulminant hepatic failure. GI hemorrhage is usually not the result of varices, as is frequently the case in patients with chronic liver failure and well-established portal hypertension, but instead is the result of stress ulceration with a significant contribution from an underlying coagulopathy. Pancreatitis is a common complication of fulminant hepatic failure. In one series, 5% of patients who died of fulminant hepatic failure and on whom an autopsy was performed had documented pancreatitis (32). The cause of this high incidence of pancreatitis is speculative but may include circulating cytokines and inflammatory cells and, possibly, intermittent systemic hypotension.

Malnutrition

A significant consequence of both acute and chronic liver disease is malnutrition. In patients with fulminant hepatic failure, malnutrition is primarily evidenced by a significant decrease in circulating protein (albumin, transferrin, prealbumin, etc.) because these patients usually have not had adequate time to significantly reduce their skeletal muscle mass (44). The body edema that sometimes accompanies fulminant hepatic failure may provide an illusion of adequate nourishment. Because previous investigations in patients with chronic liver dysfunction indicate a catabolic condition with significant protein metabolism (45), it can be assumed that such a situation is operative and possibly more severe in those patients with fulminant hepatic failure. Based on our experience, we recommend that protein supplementation be initiated in the early phases of the illness; however, close attention needs to be paid to the patient's neurologic and renal status so as to not exacerbate hepatic encephalopathy and uremia. The use of branch-chain amino acids for the promotion of protein anabolism in the treatment of hepatic encephalopathy remains controversial.

Metabolic Disorders

Disorders of glucose metabolism occur frequently in patients with liver disease. Hyperglycemia is the usual finding in those patients with liver dysfunction who are in the ICU. However, hypoglycemia can occur, especially in those patients with end-stage fulminant hepatic failure. Because the ability to generate glucose through gluconeogenesis is one of the last functions to fail in the liver, hypoglycemia is an ominous sign of an impending terminal situation. In patients with chronic liver disease (especially those with portal

hypertension), common metabolic findings are hypokalemia and intravascular volume constriction with metabolic alkalosis. In contrast, patients with fulminant hepatic failure may develop a hyperkalemic anion-gap metabolic acidosis from both their acute liver failure and their developing renal insufficiency. Hypocalcemia will usually occur in patients with fulminant hepatic failure; however, this finding is primarily based on a low serum albumin and ionized-calcium levels are frequently normal. While serum-magnesium levels are usually depressed in patients with chronic liver disease, measurements of total serum magnesium are frequently within normal limits or sometimes elevated (possibly due to impending renal failure) in the patients with fulminant hepatic failure (44).

Renal Dysfunction and Failure

Renal dysfunction occurs commonly in patients with liver disease. Renal dysfunction and failure occur by primarily two mechanisms: acute tubular necrosis and the hepatorenal syndrome. Patients with chronic end-stage liver disease and those patients with fulminant hepatic failure may be intermittently hypotensive, on nephrotoxic antibiotics, and infected with microorganisms, all of which can predispose them to the development of acute tubular necrosis. Acute tubular necrosis can occur at any time during the course of the liver disease.

Development of the hepatorenal syndrome heralds a terminal condition and is, thus, frequently associated with end-stage fulminant hepatic failure. The hepatorenal syndrome is characterized by overly avid sodium retention, with extremely low urine sodium and fractional excretion of sodium (less than 1%). Once the hepatorenal syndrome occurs, the use of intravascular volume expansion and renal-dose dopamine hydrochloride may temporarily improve the situation; however, the onset of hepatorenal syndrome is an indication that definitive therapy (e.g., liver transplantation) will need to be performed if the patient is to survive. The renal dysfunction of hepatorenal syndrome will resolve with restoration of liver function (46).

Treatment

Patients with fulminant hepatic failure should be admitted to a hospital with a liver-transplant program. The person obtaining the history should attempt to elucidate risk factors consistent with common etiologies of fulminant hepatic failure, as outlined earlier in the chapter. Physical examination, in addition to looking for stigmata of chronic liver disease, should attempt to discern signs of involvement of the central nervous, pulmonary, cardiovascular, and renal systems, as well signs of coagulopathy and malnutrition. Routine laboratory assessment should include electrolytes, complete blood count (CBC), liver-function tests, coagulation screen, arterial blood gas (ABG) analysis, viral hepatitis serology, copper studies, uric acid, glucose, blood urea nitrogen or creatinine levels, drug and toxicology screens, and ammonia levels. Although an elevated ammonia level is expected, the degree of elevation does not correlate with the degree of encephalopathy (47).

The patient should receive vitamin-K supplementation (10 mg p.o. for 3 days), gastric-ulcer prophylaxis, and blood-product transfusion designed to minimize bleeding complications.

The use of sedative medications should be minimized, and appropriate treatment for certain etiologic factors of fulminant hepatic failure should be administered (e.g., n-acetylcysteine, acyclovir sodium, interferon-α, anti-HBsIg). Some evidence exists that hepatic encephalopathy is enhanced by GABA-receptor stimulation. Flumazenil, a benzodiazepine and GABA antagonist, has been used with success in providing temporary (on the order of minutes to hours)

improvement in mental status (48, 49). Flumazenil administration may be useful as a diagnostic and prognostic tool; in Bansky et al.'s report (49), none of the patients who died within 48 hours of the flumazenil challenge had improved with flumazenil use. There is only one case report (50) of long-term use of flumazenil in the management of hepatic encephalopathy.

Encephalopathy can be graded to aid in a therapeutic plan (see Table 2) (32, 51). With the development of Grade 2 hepatic encephalopathy, the patient should be transferred to an ICU. A baseline head CT scan should be obtained, and lactulose should be administered orally, along with the initiation of either parenteral or enteral nutrition with close monitoring of plasma glucose. The patient should be pan-cultured, and consideration should be given to the initiation of selective bowel decontamination. Renal function should be monitored (serum urea, creatinine, and urine output); volume status optimized with monitoring via a central venous pressure or pulmonary artery catheter if necessary; and dialysis initiated in those patients with renal failure who are liver-transplant candidates. Dopamine given at renal doses (1-3 $\mu g \cdot kg^{-1} \cdot min^{-1}$) may be helpful in supporting renal function.

If Grade-3 hepatic encephalopathy is reached, the patient should be intubated and hyperventilated ($PaCO_2$, 25-30 mmHg), the head placed in a midposition and elevated 30^0, and the neurosurgical service contacted for ICP-monitor placement. The epidural placement of a fiberoptic ICP monitor is the current recommendation. Following placement of an ICP monitor, efforts should be made to maintain the ICP below 20 mmHg and cerebral-perfusion pressure above 60 mmHg. If renal failure is not present, the order of treatment of intracranial hypertension is hyperventilation, followed by diuretic therapy (mannitol and furosemide), followed by administration of sodium pentothal, until acceptable ICP and cerbral perfusion pressures are achieved. If renal failure is present, one would omit the diuretic therapy from the algorithm. Daily head CT scans should be obtained when feasible.

If the patient achieves Grade-4 hepatic encephalopahty (coma, unresponsive to pain), efforts should be made to detect evidence of irreversible brain injury. These measures can include ICP monitoring, head CT scanning, electro-encephalography, radiolabeled cerebral-flow studies, and cerebral angiography.

Mediator Therapy

The failing liver resides in an environment of circulating soluble mediators, including cytokines; arachidonic-acid metabolites; and products of cytokine-activated cells, such as nitric oxide (23, 52, 53). Further hepatotoxicity results when these mediators are improperly cleared. Therapy for patients in whom these inflammatory substances are present in increased amounts is in an experimental phase but, in the future, may consist of immunotherapies and receptor blockers against lipopolysaccharide, tumor necrosis factor, interleukin 1, and neutrophil surface antigens (54-57). The inhibition of nitric-oxide synthesis with arginine analogs may assist in the treatment of the failing liver or may possibly ameliorate the damaged liver's effect on other organ systems (58). Selective bowel decontamination can be viewed as an indirect form of mediator therapy because the resultant reduction in bacterial translocation can reduce circulating endotoxemia (43).

Liver-Support Devices

Many methods of liver support besides liver transplantation are currently under investigation. Hepatocyte transplantation (59), extracorporeal liver-assist devices (60-62), and plasma exchange are now undergoing, or will soon undergo, trials to determine

their merit in extending patient survival through a period of hepatic dysfunction while regeneration or donor identification for transplantation occurs. Auxiliary transplantation (transplanting a portion of an allograft with the recipient's liver in situ) is a surgical means of providing temporary support to a patient allowing time for hepatic regeneration in hopes of avoiding orthotopic liver transplantation (63). Xenografting (the use of nonhuman organs) has been attempted with some temporary success (64). Genetic strategies are being explored that would alter the immunogenic character of laboratory animals to make them more useful for temporary or permanent transplantation.

Liver Transplantation

Orthotopic liver transplantation remains the treatment of choice for long-term survival in most patients with fulminant hepatic failure. The patient survival in recently published series, where liver transplantation was an option, ranged from 56% to 62% (30, 65-67). Survival figures from the pretransplantation era were in the range of 6% to 19% (27, 68-70). Thus, in spite of the absence of a randomized controlled trial between liver transplantation and conservative medical management for patients with fulminant hepatic failure, current data suggest that liver transplantation has made an important contribution in the care of patients with this illness.

The dilemma for the clinician caring for a patient with fulminant hepatic failure is not only if, but when, to transplant. The goal is to allow enough time for liver regeneration while not waiting so long as to significantly compromise the patient's clinical status before transplantation. In one investigation, when liver transplantation was accomplished before the patient achieved Grade 4 hepatic encephalopathy, 91% of patients survived (32), whereas if they had achieved Grade 4 encephalopathy, the survival rate was only 25%.

A decision regarding liver transplantation must be made by a liver-transplant team after discussions with the patient, the patient's family, or the patient and family and should be based on the patient's underlying disease, stage of hepatic encephalopathy, and whether or not the patient is beginning to fulfill O'Grady's criterion for fulminant hepatic failure (33). O'Grady showed that in patients with acetaminophen-induced hepatic failure, a poor prognosis with medical therapy alone was associated with an arterial pH less than 7.30, a prothrombin time (PT) greater than 100 seconds, or a creatinine level greater than 300 μmol/L. With other causes of fulminant hepatic failure, poor medical outcome was associated with a patient age less than 11 years or greater than 40 years, a jaundice to encephalopathy interval greater than 7 days, a serum bilirubin level greater than 300 μmol/L, or a PT greater than 50 seconds.

Summary

Patients with fulminant hepatic failure provide the intensivist with the challenge of caring for a patient who, in the best of circumstances, will have a 56% to 62% chance for survival. Although some similarities exist between fulminant hepatic failure and chronic liver disease, the differences are extremely important to understand. The goal of the intensivist is to diligently support the failing liver and meticulously care for other organ systems at risk for failure. The patient would benefit most from a situation that would allow a proper amount of time for liver regeneration to occur (possibly with the help of liver-support devices) and yet not permit so much time as to allow for the development of multiple organ system failure, which would greatly complicate liver transplantation. Orthotopic liver transplantation remains the best opportunity for long-term survival for most patients with fulminant hepatic failure.

Eye to the Future

Future developments may follow a number of pathways. Agents to block the effects of the toxic mediators liberated (or not cleared) by the failing liver may help to stabilize the multiorgan dysfunction seen in these patients. Liver-support devices, analogous to hemodialysis currently used for renal failure, may act as a bridge for the patient with fulminant hepatic failure who is awaiting either a donor organ or regeneration and recovery of the dysfunctional liver.

Key Points

Fulminant hepatic failure is a catastrophic condition that progresses more quickly and virulently than does an exacerbation of chronic hepatic dysfunction.

Fulminant hepatic failure is a multiorgan disease and, in addition to the hepatic system, may involve the cardiovascular, renal, pulmonary, neurologic, and hematologic systems.

Intracranial hypertension is a frequent cause of morbidity and mortality.

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Management of Acute Poisoning - Current Guidelines

Dr. Praveen Aggarwal

Additional Professor, Division of Emergency Medicine, AIIMS, New Delhi

Poisoning is a major problem all over the world. The exact incidence of poisoning-related mortality in India is not known, but it is estimated to be 5.7 per lakh in 1994. Kerosene oil poisoning is the commonest poisoning in children while, in adults, aluminium phosphide and insecticides are most often involved in poisoning cases particularly in north India. Knowledge of this pattern is helpful in making an early diagnosis and instituting proper treatment.

In order to reduce poisoning-related morbidity and mortality, five complementary steps are important. These are:

1. Resuscitation and Initial Stabilization
2. Diagnosis of type of poison by history, examination and laboratory investigations.
3. Nonspecific therapy to reduce the levels of toxin in the body
4. Specific therapy to reduce the toxic effects on the body
5. Supportive care to support the functions of vital organs.

Resuscitation and Initial Stabilization

On arrival of a patient with poisoning, the initial priorities are the maintenance of airway, breathing and circulation. If the patient has altered level of consciousness, his cervical spine must be immobilized till an injury can be ruled out. In presence of respiratory inadequacy, endotracheal intubation is required. Hypotension in poisoned patients is most often due to loss of fluids or toxin-induced vasodilatation. Hence, crystalloids are the first choice of treatment of hypotension. Before infusing fluids, blood should be withdrawn.

After initial resuscitation, all patients with altered sensorium should receive a 'cocktail' of 50% dextrose, naloxone and thiamine. However, recently, empiric administration of dextrose has been questioned. Administration of glucose to normoglycemic or hyperglycemic animals during cardiopulmonary resuscitation has been shown to be associated with worse neurologic outcome compared to animals who did not receive glucose. Similar conclusions have been made in human studies involving stroke and head injury victims. At present, it is recommended to estimate the blood sugar using a reliable bedside test and to administer dextrose only if the blood sugar is below 80 mg/dl.

Another component of the 'cocktail' recommended in patients with altered mental status is naloxone. It is able to rapidly counteract the sedation and respiratory depression induced by opiates. The dose is 2 mg in all age groups. However, if the patient may be an opioid addict and he is not apnoeic, the initial dose may be reduced to avoid withdrawal features. Naloxone can occasionally produce side effects in the form of hypertension, pulmonary oedema, arrhythmias, seizures and cardiac arrest. It can also precipitate the withdrawal reaction.

Diagnosis of Type of Toxin

History: The history should be elicited from the patient as well as his relatives. Occupational history and availability of potential poisons at home should also be asked for.

Examination: The examination should focus on the vitals (pulse, blood pressure, temperature, respiration), neurologic functions, cardiopulmonary and abdominal status, and breath odour. Based on the examination

findings, it may be possible to identify the type of poison involved (Table 1).

Laboratory Investigations: A few simple bedside tests are helpful in diagnosing the chemical ingested. A pinkish colour of urine occurs in phenothiazine intoxication, as well as in myoglobinuria and hemoglobinuria. Chocolate-coloured blood is indicative of methemoglobinemia. Presence of oxalate crystals in urine is typical of ethylene glycol ingestion. Ketonuria without any metabolic change occurs in isopropyl alcohol and acetone intoxication while ketonuria with metabolic acidosis is suggestive of salicylates poisoning.

Besides simple laboratory tests, abdominal X-rays may be useful in diagnosing certain radio-opaque toxins which include chloral hydrate, heavy metals, iron, iodides, phenothiazines, sustained-releases preparations and solvents (chloroform, carbon tetrachloride). However, one must not exclude a poisoning on the basis of absence of radio-opaque density on X-ray.

Table 1. Clinical Features and Associated Poisons

Clinical Features	Poisons
Odour of Breath	Chloroform, Ethanol, Cyanide Arsenic, Organophosphates, Phosphorus, Kerosene
Hypertension with tachycardia	Amphetamines, Cocaine, Marijuana, LSD, MAO inhibitors, Nicotine, Sedative or alcohol withdrawal syndromes, Antihistamines, Antipsychotics, Tricyclic antidepressants
Hypotension with bradycardia	Antidepressants (severe cases), Barbiturates, Benzodiazepines, Nicotine, Organophosphates
Hypotension with tachycardia	Aluminium phosphide, Antipsychotics, Caffeine, Cyanide, Disulfiram-ethanol interaction, Tricyclic antidepressants
Hyperthermia	Amoxapine, Amphetamines, Antidepressants, Cocaine, Lithium, Salicylates, LSD, MAO inhibitors, Phencyclidine, Anticholinergics, Antihistamines, Antipsychotic agents.
Hypothermia	Antidepressants, Ethanol, Benzodiazepines, Narcotics, Barbiturates, Phenothiazines
Tachypnoea	Amphetamines, Atropine, Cocaine, Salicylates, Carbon monoxide, Cyanide, Metabolic acidosis
Bradypnoea	Antidepressants, Antipsychotic agents, Barbiturates, Ethanol, Benzodiazepines, Chlorinated hydrocarbons, Narcotics, Nicotine, Organophosphates, Cobra bites
Altered sensorium	Antidepressants, Antipsychotics, Antihistamines, Atropine, Organophosphates, Barbiturates, Carbon monoxide, Benzodiazepines, Ethanol, Narcotics, Cyanide, Lithium
Seizures	Antidepressants, Antipsychotics, Antihistamines, Cocaine, Chlorinated hydrocarbons, Organophosphates, Lead and other heavy metals, Lithium, Narcotics, Amphetamines
Miosis	Barbiturates, Phenothiazines, Ethanol, Narcotics, Nicotine, Organophosphates
Mydriasis	Amphetamines, Caffeine, Cocaine, LSD, MAO inhibitors, Nicotine, Antidepressants, Antihistamines, Atropine
Cyanosis	Methaemoglobinaemia-inducing agents, Terminal stage

Non-specific Treatment

The next step in the management of a poisoned patient is to remove the unabsorbed poison from the gut and increase the excretion of absorbed poison from the body. The first objective can be achieved by several means including induction of emesis, gastric lavage, and use of activated charcoal and cathartics.

A. Removing Unabsorbed Poison from gut

Before performing a procedure for gastric emptying, one has to consider (i) whether the ingestion is potentially dangerous, (ii) can the procedure remove a significant amount of toxin, and (iii) whether the benefits of a procedure outweigh its risks. If the patient has ingested a non-toxic agent, non-toxic dose of a toxic agent, or if he is free of symptoms despite passage of time during which the toxin is known to produce features of toxicity, gastric emptying is unnecessary. However, if the patient has ingested a high-risk toxin (cyanide, paracetamol), gastric emptying is indicated even if he is asymptomatic. Gastric emptying is also not indicated if the patient had prior repeated vomiting, the toxin is absorbed rapidly, or patient presents late after ingestion. However, some toxins (antidepressants, phenothiazines, salicylates, opioids, phenobarbital and anticholinergics) delay gastric emptying. Gastric emptying is also delayed in comatose patients. It is also delayed if the toxin forms a mass in the stomach. In these situations, a delayed gastric emptying may have theoretical benefits but the data does not support it. If the risks of a procedure outweigh the possible benefits, it should be avoided (e.g., ingestion of volatile hydrocarbons, caustics).

1. *Syrup of ipecac*: Syrup of ipecac is used to induce emesis with the intention to remove the poison from the stomach. It was recommended in all patients with poisoning who present within one hour of ingestion. The value of syrup of ipecac has been investigated in animal and human studies. The amount of recovery of toxin has been highly variable and after one hour, the amount is insignificant. Thus, presently, ipecac may be considered in an alert conscious patient who has ingested a potentially toxin amount of a poison within the last one hour. It should be avoided in ingestions of hydrocarbons and corrosives. It is not available in India.
2. *Gastric lavage:* For orogastric lavage, the patient should be in a left lateral position with the head lower than the feet so as to avoid aspiration. In adults and children above 6 years, tap water is sufficient for lavage. However, children below the age of 6 years should be lavaged using saline at body temperature. A lavage is contraindicated following ingestion of strong caustics, non-toxic agents and volatile hydrocarbons.

Gastric lavage is routinely performed in all patients with poisoning. However, the data from studies does not support the routine use of lavage in all patients with poisoning. No animal studies has shown substantial drug recovery, particularly if lavage was delayed for 60 minutes. Clinical studies have also not confirmed the benefit of gastric lavage alone even when it was performed within 60 minutes of ingestion. Further, there is a possibility of enhancement of drug absorption as lavage may propel the poison into small intestine. The utility of gastric lavage has also been studied in comatose patients. It was concluded that routine lavage in unconscious patients should be regarded as potentially dangerous in all cases and of no value in most.

Based on the experimental and clinical studies, gastric lavage should not be considered unless the patient has ingested a potentially life-threatening amount of poison and the lavage can be undertaken within 60 minutes of ingestion.

3. *Catharthics:* Cathartics have been used for several years with the hope of increasing the excretion of the toxins from the gut. Oil-based cathartics are contraindicated as they increase the absorption of several toxins. To prevent complications, repetitive doses of these agents should be avoided. These complications include electrolyte imbalance, dehydration, and in case of sorbitol, abdominal distension. Cathartics are contraindicated in presence of ileus, intestinal obstruction, renal failure, hypotension, severe diarrhoea and abdominal trauma. However, there is no data to support their efficacy and their use cannot be recommended at present.

4. *Activated charcoal:* Use of activated charcoal has revolutionized the treatment of poisoning. The usual dose is 1 g/kg body wt. or 10 parts of charcoal for every one part of toxin, whichever is greater. Activated charcoal is contraindicated in patients with unprotected airway and caustic ingestion. Although not effective in adsorbing lithium, iron, DDT, methanol, ethanol, metals and hydrocarbons, it is not contraindicated in these ingestions.

   Based on the results of the volunteer studies, activated charcoal is likely to be beneficial if administered within 60 minutes of toxin ingestion. Therefore, in such situations, it may be considered if a patient has ingested a potentially toxic amount of a poison. It may also be considered beyond 1 hour but the data is insufficient to support or exclude its use. Unfortunately, activated charcoal is not available in India.

5. *Whole bowel irrigation:* In this method, isotonic solution of polyethylene glycol-electrolytes is administered orally in a dose of 2 litres/hour in adults and 0.5 litre/hour in children. The procedure is continued for 4-6 hours or till the rectal effluent is clear. The components of this solution are not absorbed through the intestines. At present, there are no established indications for the use of whole bowel irrigation. Based on the experimental studies, WBI is an option for potentially toxic ingestions of sustained release or enteric coated drugs. It has a theoretical value in the management of iron ingestion and ingestion of drug packets. It is also of theoretical value if the toxin is not adsorbed by activated charcoal.

B. Enhancement of Excretion of Toxin

Once the absorption of a toxin has been reduced by various methods, the next logical step is to enhance the elimination of already absorbed toxin from the body. Important methods for this purpose are forced diuresis with alteration in urinary pH, multiple doses of charcoal, peritoneal and hemodialysis, hemoperfusion, hemofiltration and exchange transfusion.

1. *Forced alkaline diuresis:* One of the commonly used methods to increase the elimination of a toxin is forced diuresis with alteration in urine pH. Alkaline diuresis is useful in salicylates, phenobarbital and lithium intoxication. For alkaline diuresis, 5% dextrose in half normal saline containing 20-35 meq/L of bicarbonate is administered at rate so as to produce a urine output of 3-6 ml/kg/hour and a urine pH 7.5-8.5. Diuretics are often needed to maintain high urine flows. To prevent hypokalaemia, potassium should be added in every second or third bottle. During forced alkaline diuresis, the vitals of the patient along with input/output, electrolytes and acid-base status should be closely monitored. This procedure is contraindicated in patients with shock, hypotension, renal failure and congestive heart failure.

2. *Multiple-dose activated charcoal:* Recently, multiple doses of activated charcoal have been recommended in treating certain poisoning. Because of multiple doses, free charcoal is available in the intestines to bind any toxin which has significant enterohepatic circulation. Further, free toxin in the blood tends to diffuse out of the blood into the intestines where it binds the charcoal, thereby maintaining the concentration of free toxin in the intestines near zero. This is termed "gastrointestinal dialysis". Depending upon the

severity of poisoning, the doses are: 0.5-1 g/kg body weight every 1-4 hours. Multiple doses of charcoal are indicated in following conditions: (a) If the toxin has a long half life; (b) if the toxin has a significant enterohepatic circulation (digoxin, phenobarbital, theophylline); (c) if continuous release of toxin occurs from a sustained-release preparation; (d) if a toxin forms a mass in the gut which is a source of continuous release of toxin; and (e) if the ingestion is too massive to be effectively adsorbed by a single dose of charcoal.

3. *Dialysis:* Peritoneal and hemodialysis are useful for water-soluble compounds of low molecular weight. The chemical should have a low volume of distribution and should have a low serum protein-binding. Also, the chemical should not be irreversibly bound to the tissues. These are useful in ethanol, methanol, salicylate, theophylline, ethylene glycol, phenobarbital and lithium intoxications. Peritoneal dialysis is a slow process and it should not be used if facilities for hemodialysis are available.

Specific Therapy

If the toxin can be identified, specific therapy using antidotes should be administered. However, antidotes are available only for a few toxins only. Further, the availability of various antidotes is limited in India. Table 2 lists some of the commonly used antidotes along with their dosages.

Supportive Therapy

Since the antidotes are available only for a few toxins, the treatment of most of the cases with poisoning is largely supportive. The aim of the supportive treatment is to preserve the vital organ functions till poison is eliminated from the body and the patient resumes normal physiological functions. Therefore, functions of central nervous system, cardiopulmonary system should be supported with proper care for coma, seizures, hypotension, arrhythmias, hypoxia, and acute renal failure. The fluid, electrolyte and acid-base status should be closely monitored in all patients.

Table 2. Antidotes and their use

Antidote	Poison	Administration
Atropine	Cholinesterase inhibitors	Initially, administer 2-4 mg for adults and 0.05 mg/kg for children. Repeat every 5-15 minutes until there is cessation of oral and tracheal secretions. Then lower the dose and give at less frequent intervals to maintain atropinization for 48-72 hrs
Pralidoxime	Organophosphates	1-2 gm (25-40 mg/kg in children) IV over 10-20 minutes. Repeated every 4-8 hours
Naloxone	Opiates	See under 'Resuscitation'
Methylene blue	Methaemoglobinemia	1-2 mg/kg body weight as a 1% solution to be given slowly over 5 minutes intravenously. May be repeated after 1 hour
Ethanol	Methanol, Ethylene glycol	Loading dose is 0.75 g/kg which is followed by maintenance dose of 0.1 g/kg/hr
Deferoxamine	Iron	90 mg/kg (upto 1 gm) i.m. followed by 90 mg/kg (upto 1 gm) every 4-12 hours. If hypotension is present, give intravenously at a rate not more than 15 mg/kg/hour.
Snake antivenin	Snake bites	Dose varies with the species of snake and the severity of envenomation

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Organophosphate and Organocarbamate Poisoning - Controversies in Management

Dr. Mabel Vasnaik

Associate Prof. and Head, Department of Emergency Medicine, St. John's Medical College, Bangalore

Every Emergency Department has a large patient load coming with the history of insecticide poisoning, majority of which are organophosphates and organocarbamates.

Common organophosphates are quinalophos, mevinphos, monocrotophos, chlorpyrifos, bromophos, dichlorvos, methylparathion, malathion, parathion, fenthion, phosphomidon, phenitrothion, dimethoate, phorate, methyldemeton,and diazinon.

Common carbamates are bendiocarb, carbaryl, carbosulphan, methomyl, propoxur and aldicarb.

In order to understand the controversies surrounding the management of these cases we have to first understand the pathophysiology and pharmacokinetics of these drugs.

Pathophysiology

These drugs are cholinesterase inhibitors. Acetylcholine is the neurotransmitter at pre and post ganglionic parasympathetic synapses, sympathetic preganglionic synapses, and at the neuromuscular junction.

When an action potential stimulates the nerve terminal, specific quanta of acetylcholine are released into the synapse. This acetylcholine is immediately hydrolysed by acetylcholinesterase to choline and acetic acid. The turnover time for this reaction is about 150 microseconds.

Organophosphates and organocarbamates bind to the acetylcholinesterase molecule at it's active amino acid, serine and phosphorylate or carbamoylate it respectively. The resultant conjugate is much more stable than the acetylcholine -acetylcholinesterase conjugate.

Carbamoylated enzyme spontaneously degrades in minutes or hours so that the enzyme site is eventually regenerated. Hence carbamate poisoning spontaneously reverts in 24 to48 hours.

However, phosphorylated enzymes degrade over days to weeks, making the acetylcholinesterase essentially inactive.

In order for the physiologic enzyme activity to return new enzyme must be generated or an antidote given.

Pharmacokinetics

The organophosphate insecticides are a closely related family of chemicals varying in structure. The various groups attached to the different sites of the phosphorus moiety influence the relative tightness of the bond to acetylcholinesterase, the time for hydrolysis, the potency and the latency of onset of symptoms.

The pharmacologic and toxicologic effects of organophosphates and carbamates are due predominantly to inhibition of acetylcholinesterase and accumulation of acetylcholine at the synapse. The excess acetylcholine paralyses the cholinergic synaptic transmission within the CNS, somatic nerves, autonomic ganglia, parasympathetic nerve endings and some sympathetic nerve endings like in the sweat glands.

SIGNS AND SYMPTOMS

Muscarinic Effects

Salivation, Lacrimation, Urination, Diarrhea, Gastrointestinal distress, Emesis, Miosis, Bronchorrhea & Bronchospasm, Bradycardia, Sweating excessively.

Nicotinic Effects

Muscle fasciculations, cramping, weakness, Diaphragmatic paralysis, respiratory failure and Areflexia.

CNS effects

Restlessness, headache, emotional lability, drowsines, tremors, slurred speech, confusion, delirium, general weakness, ataxia and seizures.

Parenteral injection of organophosphates can cause necrosis and abscesses at the site of injection and a severe poisoning reaction of late onset.(32)

Diagnosis

Diagnosis is made on

1. History of ingestion of the compound.
2. Signs and symptoms
3. Improvement after oxime therapy and atropine
4. Inhibition of cholinesterase activity

Depression of cholinesterase is an essential finding. However, since a normal cholinesterase level is based on population estimates, a single isolated level neither confirms nor excludes exposure. Neonates and infants have baseline cholinesterase activity that is lower than adults.

Ideally, diagnosis is based on a drop of 50 % from baseline cholinesterase values. Since most people do not have a known baseline, the diagnosis can be confirmed by a progressive increase in the cholinesterase values.

Mild poisoning is defined as a cholinesterase activity of 20 to 50%, moderate 10 to 20% and severe less than 10% of normal enzyme activity.

Table 1. Interpretation of Cholinesterase Levels

	Red Blood Cell Cholinesterase	Plasma Cholinesterase
Advantage	Better reflection of synaptic inhibition	Easier to assay, declines faster
Site	CNS gray matter, RBC, motor end-plate	CNS white matter, plasma, liver, pancreas, heart
Regeneration (untreated)	1%/day	25-30% in first 7-10 days
Normalization (untreated)	35-49 days	28-42 days
Use	Unsuspected prior exposure with elevated plasma cholinesterase	Acute exposure
False depression	Pernicious anemia, hemoglobinopathies, antimalarial treatment, oxalate blood tubes	Liver dysfunction (cirrhosis), malnutrition, Hypersensitivity reactions, drugs (succinylcholine, codeine, morphine), pregnancy, genetic deficiency

Treatment Aims

1. Reversal of the synaptic biochemical abnormalities.
2. Reversal of cholinesterase blockade.

This is achieved by administering a sufficient quantity of two complementary medications.

A) Atropine

A tertiary amine, it is a competitive antagonist of acetylcholine at the muscarinic postsynaptic membrane and in the CNS.

In symptomatic poisoned adults, atropine is given as 1 to 2mg IV/IM (0.02 - 0.05mg/kg in children). If there is no effect the dose is doubled every 5 to 10 minutes until muscarinic symptoms are relieved. In massive exposures hundreds of milligrams of atropine may be needed. Atropine can also be given via a continuous intravenous infusion.

The patient should be oxygenated prior to atropine administration in order to prevent ventricular dysrhythmias associated with hypoxia.

Freeman et al (2) concluded in a statistically significant study that patients who received heavy doses of atropine survived more frequently than those who received inadequate doses or none at all.

Senanayake et al (3) in their review on organophosphate insecticide poisoning observed that an atropine infusion was more effective than intermittent bolus doses and inadequate atropinisation was one of the main causes of pneumonia. An early and adequate control of secretions prevents pneumonia and decreases the incidence of respiratory failure.

Sunder Ram et al (4) in 1991 studied the effect of continuous high dose atropine in the treatment of 45 cases of OP poisoning. They observed a statistically significant reduction in mortality compared to cases treated earlier with lower dosage regimes.

Singh et al (5) from PGIMER, Chandigarh felt that aggressive atropinisation increased survival in cases of OP poisoning. The mean dose of atropine used on day 1 was 178.9mg (range 60-480mg) and then gradually reduced. Mean duration of treatment with atropine was 9.6 days.

The end point of anticholinergic treatment is clearing of the secretions from the tracheobronchial tree and drying of most secretions. Pupillary dilatation is an early response to atropine, but it is not a therapeutic end point. Tachycardia is not a contraindication to atropine.

Once signs of adequate atropinisation occur, the dose should be adjusted to maintain this effect for at least 24 hrs, and carefully withdrawn once the patient is adequately stabilized. Atropine should be restarted at the first sign of recurrence of cholinergic symptoms.

Glycopyrrolate (0.05mg/kg), a quartenary ammonium compound does not penetrate the CNS, and may be substituted for atropine in a patient with a clear sensorium, with no evidence of central toxicity, thus eliminating the potential for a central anticholinergic syndrome seen with atropine. There is also less tachycardia and a better control of endobronchial secretions and hence a lower incidence of respiratory tract infections.

Choi et al (6) describe a case of malathion ingestion, who despite clinical signs of adequate atropinisation continued to have profuse bronchorrhea which resolved with glycopyrrolate.

B) PRALIDOXIME

(2 hydroxy imino methyl -1 - methyl pyridinium chloride, 2-PAM) and other oximes function by a nucleophilic attack on the phosphorylated enzyme, removing the bulky phosphate moiety and completely restoring normal acetylcholinesterase activity. They may also prevent continued toxicity by scavenging the remaining organophosphate molecules.

PAM should be administered soon after diagnosis is made. Once the phosphorylated enzyme has aged, the phosphate group becomes irreversibly bound to the enzyme and oxime therapy is no longer as effective. However, review of literature shows that restriction of the use of PAM to 48 hrs is unjustified and may have deleterious effects. Decrease in muscle weakness and fasciculations occurs within 10 to 40 minutes of PAM. However regeneration of cholinesterase is dependent on plasma concentrations of the organophosphate compound. Delayed clinical manifestations occur with fat-soluble organophosphates like fenthion, chlorfenthion and parathion, and multiple doses of PAM are necessary.

PAM is not equally antagonistic to all organophosphates. It has a greater effect on diethyl compounds like parathion, mevinphos and diazinon compared to dimethoxy compounds like malathion and methyldemeton.

PAM is administered via a bolus injection 20-40mg/kg dissolved in 0.9% Normal saline and infused over 30 minutes. This dose can be repeated every 6 to 12 hours. However, plasma levels are not maintained as the elimination half-life of PAM is 1.2 hours.

Namba et al (7) showed that a continuous infusion is more effective than multiple single injections.

Serious intoxication may require a continuous infusion of 500 mg/ hr in adults and 9 to19 mg/kg/hr in children. PAM is also effective IM and PO but IV infusion is the preferred route as it has a rapid onset of action and reliable kinetics. Side effects of rapid IV bolus are hypotension, dizziness and blurred vision. Cardiac and respiratory arrests have been known to occur.

PAM has a high therapeutic index and several studies that have used large doses over prolonged periods have not shown any toxicity. Adverse effects of PAM are evident only with plasma levels greater than 400 ug/ml. Several studies have been done to find the minimum serum level necessary for therapeutic efficacy of PAM and based on animal data a serum level of 4ug/ml is suggested as the minimum therapeutic threshold.

Willems et al (8) infused 4.42 mg/kg bolus over 30 minutes followed by 2.14 mg/kg/hr. Medicis et al (9) infused 4 mg/kg bolus over 15 minutes. followed by 3.2 mg/kg/hr. Both groups achieved serum pralidoxime levels greater than 4 ug/ml.

However, organophosphate residues that are present may bind the circulating free PAM and lower it's serum concentration. PAM should therefore be continued as long as nicotinic symptoms persist. CNS effects of PAM are controversial as it is a quartenary nitrogen compound and does not cross the blood brain barrier.

Worek F et al (10) in their study on reappraisals of indications and limitations of oxime therapy in OP poisoning suggested that oximes be administered by a continuous infusion following an initial bolus dose as long as reactivation can be expected and until permanent clinical improvement is achieved.

Tush et al(11), Futagami et al(12), Kipling et al (13), Grob et al (14) and Namba et al (7) in their studies recommended both a continuous PAM and atropine infusion in OP poisoning.

Farrar H.C. et al (15) in their study advocate the use of a continuous infusion of PAM for treatment of OP poisoning in children.

De Silva et al (16) in their study on 45 cases of OP poisoning found no difference in the outcome of cases with or without the use of PAM. They used a dose of 1gm of PAM a day for 5 days.

Johnson et al (17) felt that there is substantial morbidity and mortality in cases of OP poisoning treated without oximes. They felt that the dose of PAM used by De Silva et al (16) was inadequate.

Although a review of literature by and large favours an adequate dosage of oximes in the treatment of OP poisoning, two studies done nearer home do not advocate the use of PAMs.

Samuel J et al (18) in a randomised double blind, controlled clinical trial evaluated two treatment regimes of PAM in 72 patients. One group got 1 gm of PAM bolus on admission, while the other group was given a 6 gm infusion on the first day, 3gms on the 2nd, 2 gms on the 3rd and 1gm on the 4th day. They found that there was a greater incidence of intermediate syndrome (IMS) and an increased ventilatory requirement in the high dose group. However, they also found that those getting PAM within the first 12hrs of ingestion showed a decreased incidence of IMS and ventilatory requirement.

Subsequently, they did another study (19) on 110 organophosphate poisoned patients. One group was given a placebo, whereas the other group was given 12 gms of PAM over 3 days. They found that there was higher mortality rate, ventilatory requirement, incidence of IMS and infections in the group that received PAM.

The other OXIMES are monoisonitrosoacetone (MINA), diacetyl monooxime (DAM), trimedoxime, bisaldoxime and obidoxime (Toxogonin).

Obidoxime is effective in smaller doses 250mg IV/IM as a slow bolus or infusion. It is more potent and more toxic than PAM.

Balali et al (20) in their study on 63 patients of OP poisoning found that high doses of obidoxime (8mg/kg followed by 2mg/kg/hr) were found to be hepatotoxic and should be avoided.

Thiermann et al (21) in their study on modern strategies in the therapy of OP poisoning concluded that no IMS or signs of permanent hepatic dysfunction were found in 12 patients treated with obidoxime and that the often cited ineffectiveness of oximes may be due to an inappropriate dosing.

Atropine and PAM given together are synergistic against the signs and symptoms of cholinesterase inhibition, thus improving the outcome in these patients.

Other Medications

1) The addition of diazepam to atropine and PAM decreases cardiac and brain morphologic damage resulting from OP seizures and adequate doses of benzodiozepines should precede the use of barbiturates. The use of phenytoin is not recommended because of its membrane stabilizing and autonomic effects.

2) Many opioids are partially metabolised by the pseudocholinesterase pathway and so their use for sedation or pulmonary edema may worsen CNS manifestations and respiratory depression.

3) Drugs metabolised via plasma cholinesterases like succinylcholine, mivacurium, and esmolol have an increased duration of action in these patients and should be avoided. Non depolarising muscle relaxants rely on the acetylcholine effect at the neuromuscular junction and so, patients may be resistant to their effects.

Organocarbamates

Their effect is more transient and most acetylcholinesterase spontaneously hydrolyses within 24 hours. There is a controversy over the use of PAM in treating symptomatic carbamate exposures.

Some studies have shown that in the presence of carbaryl, acetylcholinesterase inhibition may increase and atropine efficacy may decrease if 2-PAM is given.

Zweiner et al (22) in their study have observed that in carbamate poisonings oxime therapy does not contribute to recovery of patients.

Conversely Burgess et al (23) has published a report wherein a case of aldicarb poisoning with

prolonged cholinesterase inhibition showed improvement after PAM therapy.

However, a review of literature shows that most studies do not favour the use of oximes in organocarbamate poisoning. Whenever there is a doubt as to the identity of the compound, use of pralidoxime is advocated.

Other Studies

Novikova et al (24) in their study showed that *hemoperfusion* effectively removed the organophosphate from 163 cases of poisoning.

Goel et al (25) studied 103 patients and felt that need for ventilatory support was significantly more with greater time duration for institution of specific treatment, low level of sensorium and the presence of respiratory insufficiency at admission, pin point pupils, generalized fasciculations, convulsions and a higher initial atropine requirement.

Decontamination

1) The patient should be thoroughly washed with soap and water. Treating personnel should wear gloves and non permeable gowns.

2) Gastric lavage should be done within 60 minutes of ingestion. If protective reflexes are lost, patient should be intubated.

3) Activated charcoal should be given in a dose of1gm/kg.

4) In patients with ocular exposure the eyes should be well irrigated with Normal Saline, Ringer lactate or even tap water.

Delayed And Long Term Neurological Sequelae

1) Intermediate syndrome (IMS) occurs approximately 24 to 96 hours after resolution of an acute, severe, cholinergic crisis. Clinically patients develop acute respiratory paralysis, weakness in the bulbar musculature, nuchal weakness, proximal limb weakness and depressed reflexes.

A consensus from literature search appears that IMS may result from inadequate therapy with oximes. The symptom complex begins at a time when the cholinesterase function is very low and the OP compound is still detectable in the body. As blood levels of OP fall, and OP tissue redistribution occurs, the motor endplate may be rechallenged by the cholinesterase inhibitor in the presence of inadequate circulatory oximes.

PAM should be continued and adequate ventilatory support provided.

2) OP induced delayed neurotoxicity (OPIDN) or polyneuropathy (OPIDP) occur 1 to 3 weeks after exposure. Initially there is symmetric lower extremity weakness, glove and stocking paraesthesias, leg cramping and calf pain which ultimately progresses to the upper extremities. Sensory symptoms resolve over the next few months, but paralysis persists.

Mortality

Yamashita et al (26) found a 25% mortality in their cases studied. The mortality was due to a delay in discovery and transport and insufficient respiratory management.

Agents with a significant CNS penetration may cause prolonged seizures. Some organophosphates requiring hepatic metabolism for activation are directly hepatotoxic.

Recent Advances

1) Van Helden et al (27) felt that drug induced inhibition of acetylcholine release shortly after acute OP intoxication, could prevent and counteract convulsions and improve survival. Pilot experiments have found that adenosine receptor antagonists might be suitable drugs.

2) Ashani et al (28) showed that organophosphorus hydrolases can effectively hydrolyse quartenary ammonium containing OPs and will be a useful adjunct to oximes for the reactivation of OP - acetylcholinesterase complexes.

3) Lallement et al (29) studied G K - 11, an antiglutamatergic drug as additional neuroprotective therapy for OP poisoning.

4) Dihydropyridine derivative of PAM (apro-2-PAM) acts as a prodrug or drug carrier allowing passage of drug through the blood brain barrier, thus increasing the central effects of PAM.

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Assessment and Management of Psychiatric Emergencies: A Primer for Non-psychiatric Physicians

Dr. K. Srinivasan

Professor, Department of psychiatry, St. John's Medical College Hospital, Bangalore

Psychiatric emergencies present acutely with an alteration in behaviour, mood or thought, which if not rapidly attended to may result in harm to the patient or others. Psychiatric emergency services defined as a face to face evaluation of patients 24 hours a day has expanded considerably in the last two decades and has acquired a sub-specialty status. Many psychiatrically ill persons live with their families in the community because of "de-institutionalization" and development of effective community mental health systems. However, such patients due to decompensation and recurrence of symptoms often seek help from emergency services. In addition, the rapid growth of emergency medicine as a super-specialty has also contributed to a significant extent in the development of psychiatric emergency services.

Psychiatric emergencies can arise in different contexts. They can be conceptualized into five broad categories. Firstly, there are those emergencies that are a direct consequence of a psychiatric illness, such as a suicidal attempt in a depressed patient. Secondly, an underlying medical condition may be responsible for an acute behavioural disturbance, for instance, delirium in a patient with chronic renal failure. Thirdly, patients who abuse various drugs or alcohol are brought to the emergency services either for acute intoxication or for the treatment of severe drug or alcohol related withdrawal syndromes. Occasionally, a patient may manifest behavioural symptoms because of a drug-drug interaction or a drug induced psychosis for e.g., steroid related mood disorder. Finally, individuals who are otherwise psychologically normal, but victims of extreme trauma such as a sexual assault may present to the emergency medicine with acute psychological symptoms. The psychiatric emergencies can happen in different settings such as at home, in a public place, outpatient department, medical/surgical unit of a general hospital, inpatient psychiatric unit and emergency department. However, this article will focus on the presentation, assessment and management of psychiatric emergencies in an emergency department setting.

Assessment of Psychiatric Emergencies

The goals of psychiatric emergency care are relatively rapid evaluation, containment of disordered behaviour and referral to the appropriate department for further management. Towards this aim a "triage" model has been proposed. This model, essentially, consists of three stages. To begin with, the behavioural disturbance is categorized as either emergency or acute. The next step involves medical and psychiatric assessment which would then lead to the final step in the model, that of initial management and containment of the behavioural disturbance.

Medical Assessment of Patients Presenting with Psychiatric Emergencies

Physical evaluation of patients presenting with behavioural disturbances is an important initial step in the management of psychiatric emergencies. Even when there is an obvious psychiatric condition, co-morbid medical illness may be responsible for disordered behaviour. In addition, many physical diseases can present with psychiatric symptoms (Table I). The presence of abnormal vital signs increases

Table I. Common Medical Conditions Presenting with Psychiatric Symptoms

Hypothyroidism
Hyperthyroidism
Hypoglycaemia
Diabetic Keto Acidosis
Hepatic Encephalopathy
COPD
Pneumonia
Myocardial Infarction
Hypokalaemia
Hyperkalaemia
Sub-arachnoid Haemorrhage
Encephalitis
Drug Induced for e.g., Steroids

Table II. Laboratory Tests Routinely Indicated in Emergency Room Settings

Blood Urea
Serum Creatinine
Blood Glucose
Serum Electrolytes
Liver Function Tests
Total Leukocyte Count
Differential Leukocyte Count
Urine Analysis
Serum Calcium
Serum Magnesium
Drug Serum Levels
Urine Toxicology Screen
Blood Alcohol Concentration

the likelihood of an underlying medical illness being the cause for the abnormal behaviour. If a medical condition is suspected, laboratory investigations may help in arriving at a diagnosis. Certain laboratory tests are routinely indicated in an emergency room setting (Table II). With some patients who are extremely agitated or violent, a detailed physical assessment may not be possible. In such patients, initially physical restraint or sedation may be required. Sedation can be safely achieved with either lorazepam (2-4mg IM) or IV or haloperidol (5-10 mg IM or IV). Many clinicians prefer lorazepam to haloperidol, as it tends to cause fewer side effects. It should be remembered, however, that medication is ordered to achieve maximum tranquilization with minimum sedation to allow the medical evaluation to proceed to its conclusion.

Psychological Assessment

Psychological evaluation can commence once life threatening medical conditions stabilize. Patients and their families are often highly anxious in an emergency room setting. Thus, in order to allay anxiety it is better to conduct the interview in a quiet but well lit room. It helps, especially, in patients with abnormal behaviour to assign a designated doctor or a nurse for patient care during his/her stay in the emergency ward. Initial assessment must include an examination of patient's cognitive abilities (Table III). Disturbances in cognitive functions usually indicate the presence of a medical condition. Other components of psychological assessment will be covered under individual psychiatric emergencies.

Table III. Assessment of Cognitive Functions

What is your name?
Do you know where you are?
What is today's date?
What time of the day is it now?
What is the time now?
How did you get here?
I am going to give a 5 digit number. Please repeat it back to me.

Common Psychiatric Emergencies

Suicidal Patient

It is estimated that 1000 persons commit suicide each day world-wide. According to the National Crime Records Bureau of India, suicide is among the top ten causes of death in the country and the top three causes of death between the ages of 16 and 35 years. It is reported that in our country patients with suicidal attempt constitute 0.3 percentage to 1% of all patients who visit emergency departments. Patients are brought to the emergency services after having attempted suicide or threatening to commit suicide. Patients who have attempted suicide obviously require urgent medical intervention. It is useful to do a thorough body search as it may reveal certain medications or any other substances that the patient might have used to end his/her life. Plasma estimation of drug levels and urine toxicology screen is indicated where such facilities are available. In India, poisoning is the most common method employed by patients to end their lives. Emergency room physicians should be familiar with signs and symptoms of various poisons such as organophosphorous compounds, dhatura, oleander etc. Once medical condition stabilizes, patients should be referred to a psychiatrist for further assessment and management. It is essential that all patients who attempt suicide receive a complete psychiatric evaluation as a history of attempted suicide carries a considerable risk for future attempts. Forty percent of suicide attempters have made a previous attempt, while 1% of those attempting suicide will commit suicide eventually.

Patients brought to the emergency room threatening suicide must be carefully evaluated and the threat must be taken seriously. They require a high level of supervision and should be under the direct observation of nurses. In our country where there is often a shortage of nursing personnel, a relative can provide supervision. The room where the patient is kept for observation should be free of any dangerous objects or sharp instruments. Once patient's current or potential medical condition stabilizes, initial assessment must begin with inquiring about patient's specific suicidal ideation. The presence of an active intent instead of a death wish, frequent suicidal thoughts and patient's inability to switch off suicidal thoughts are important indicators for urgent intervention. It is important also to know what has prevented the patient from acting out on his/her suicidal thoughts till now? In addition, various factors that increases the risk of attempting suicide must be evaluated (Table IV). The cornerstone of treatment in a suicidal patient is to ensure adequate patient safety, serial assessment of patient's mental status and prompt initiation of treatment (Table V).Some physicians often do not feel comfortable assessing suicidal patients, while others are openly critical of such patients in that they are "weak willed" and are a "nuisance" in a busy emergency department, where doctors are "battling" to save patient's lives from serious medical conditions.

Table IV. Assessment of Risk Factors in Suicidal Patients

Demographics: age, sex, marital status
Psychiatric Status: presence of depression, alcoholism
Medical Status: presence of concurrent acute or chronic medical illness
Social Factors: employment status, living arrangements (alone or with family)
Familial Factors: family history of suicide, psychiatric illness, family environment
Past history of attempted suicide
Present suicidal ideation: active intent, frequency of suicidal thoughts, constant preoccupation with suicidal thoughts, communication of suicidal thoughts, detail plan regarding mode of suicidal attempt

Table V. Treatment of Suicidal Patient

Stabilize current medical condition

Provide adequate patient safety

- remove dangerous objects
- close supervision
- mechanical restraint or sedation if necessary
- therapeutic contracting

Serial mental status assessment

Initiation of treatment

Referral to a psychiatrist

It is important to remember that most suicidal attempts occur against the background of a significant life stress and majority of patients have treatable psychiatric illness. It is reported that 2/3 rd of patients had visited their family physicians in the month prior to their death by suicide. Thus, identification of suicidal risk and aggressive treatment of psychiatric illness is an important issue in the prevention of suicide.

Violent Patient

Many factors are responsible for violence or aggression seen in patients brought to the emergency departments. These include psychiatric disorders, brain injuries resulting in poor impulse control, certain character disorders, metabolic disturbances with accompanying delirium, alcohol intoxication and chaotic environment of the emergency department. Every emergency services must have a protocol to deal with violent patients. The main object of such a protocol is to ensure adequate safety to both, patients and the attending health professionals. These include environmental inputs such as availability of security personnel, safety alarms, a quiet well lit room with at least two exits and TV monitors. The management of violent patients include medical and psychiatric assessment to identify the underlying condition and prompt initiation of treatment. Specific measures to contain the violent behaviour are summarized in Table VI. Treatment of violent patients include verbal intervention, sedation and mechanical restraint. Medication should be preferably used after the medical needs are assessed. However, short term sedation may be required for medical and psychiatric assessment to proceed. In such cases, sedation with short acting benzodiazepine (Lorazepam 2-4 mg, I/M or I/V) may be used. Others have advocated the use of haloperidol (5-10 mg I/M or I/V) but, occasionally patient may develop drug induced akathisia that may worsen agitation or violent behaviour. Violent behaviour in psychiatric disorders are most often seen in patients who are concurrently abusing alcohol or drugs. Thus, recording a careful history of alcohol/drug use and a urine toxicology screen are useful strategies in such patients.

Table VI. Treatment Protocol For Violent Patient

Environmental Issues

- patient to be examined in a well lit, quiet room
- room should have two exits
- availability of security personnel
- TV monitor
- safety alarms
- room should have unbreakable plexiglass windows
- room should be free of dangerous objects

Non-verbal intervention

- stand by the side of the patient
- never turn your back to the patient
- keep distance from the patient
- avoid constant eye contact
- body search & patient's belongings for addictive drugs, syringes, needles, dangerous objects

Verbal intervention

- a designated person per patient
- speak in a clear voice
- be polite & calm
- patient to be told that violent behaviour is threatening others & is interfering with medical treatment

Restraint

- mechanical restraint (leather)
- chemical restraint (sedation with lorazepam or haloperidol)

History of current medication

Stabilization of medical condition

Referral to a psychiatrist for evaluation

Alcoholic Patient

There are several possible reasons for patients abusing alcohol to seek help from the emergency department (Table VII). Among these acute intoxication and delirium tremens are the two most common causes for admission to casualty services. The physical evaluation of such patients should pay particular attention to a detailed assessment of the neurological system including a fundus examination, signs and symptoms of liver dysfunction and features of nutritional deficiencies. Acute alcoholic intoxication usually does not require any specific treatment, but, aggression and violent behaviour may require sedation with a short acting benzodiazepine such as lorazepam. Resolution of alcohol intoxication follows steady state kinetics and patients may require to stay overnight in a observation ward. It is important to monitor their vital signs till patients recover from the effects of intoxication. An alcoholic patient brought to the emergency department with an altered level of sensorium should be evaluated for the presence of head injury, hepatic encephalopathy, metabolic disturbances (Hypoglycaemia, ketoacidosis) and severe alcohol withdrawal syndrome (Delirium tremens). If there is a doubt about the diagnosis, a CT brain scan should be ordered. Blood should be taken to test for liver functions, glucose, electrolytes and, where facilities are available, for blood alcohol levels. While correcting hypoglycaemia, patients should receive parenteral thiamine simultaneously to prevent the onset of Wernicke's encephalopathy. Patients with severe alcohol withdrawal syndrome would invariably require admission to an in-patient unit. Delirium tremens in an alcoholic patient is generally more common in the presence of concurrent infections or metabolic disturbances. Alcohol withdrawal syndrome requires treatment with benzodiazepines. Long acting benzodiazepines such as diazepam are preferred. However, in the presence of severe liver dysfunction, a short acting benzodiazepine like lorazepam or oxazepam is the drug of choice. Once the acute medical conditions stabilize, then patients must be referred to the psychiatrist for detoxification and treatment of alcohol abuse. Patients who refuse such consultation must nevertheless be advised about their harmful drinking pattern. Recent studies have suggested that even simple advice urging patients to cut down their level of alcohol consumption are effective.

Table VII. Presentation of Alcohol Related Problem to the Emergency Department

Alcohol intoxication
Delirium tremens
Epileptic fit
Head injury
Hepatic encephalopathy
Hypoglycaemia
Wernicke's encephalopathy

Delirious Patient

Potentially anybody can develop delirium and hence it is often referred to as "everyman's psychosis". A variety of medical conditions can result in delirium (Table VIII). If left untreated delirium is associated with a significant morbidity. The characteristic clinical features of delirium consists of an extreme degree of agitation or restlessness, global cognitive dysfunction (disturbances in attention, memory & orientation), alteration in sleep/wake cycle and perceptual distortions (illusions, hallucinations). Rarely, delirious patients can be withdrawn and hypoactive (quiet delirium). The intensity of symptoms in delirium can fluctuate a lot with patients having lucid intervals. Typically, in delirium there is a nocturnal worsening of symptoms. Patients with delirium should be cared for in a quiet, well lit room. A relative or a familiar figure should be by the patient's bedside, if possible, for it alleviates anxiety and helps in reorienting the patient. One should speak to such patients slowly and in a clear loud voice. Delirium tends to resolve once the accompanying medical condition is treated. However, due to extreme agitation they often require sedation. Haloperidol either orally or parenterally is the drug of choice. A dose of 5-10 mg is sufficient and can be repeated if necessary. Alternatively, a short acting

benzodiazepine like lorazepam or oxazepam is equally efficacious and preferred in delirium associated with hepatic encephalopathy and alcohol abuse.

Table VIII. Common Medical Conditions Associated with Delirium

1. Primary Cerebral Diseases

- Space occupying lesions of CNS
- Degenerative diseases
- Epilepsy
- CNS Infections including HIV-associated infections
- Cerebrovascular diseases
- Head injury

2. Systemic Diseases

A. Metabolic encephalopathies

- Hepatic, renal, pulmonary & pancreatic insufficiency states
- Endocrine disorders
- Hypoxia
- Fluid, electrolyte & acid-base imbalance
- Vitamin deficiencies (thiamine, nicotinic acid, folic acid, cyanocobalamin)
- Miscellaneous (porphyria, carcinoid syndrome)

B. Cerebrovascular complications of systemic diseases

- Hypertensive encephalopathy
- Rheumatoid vasculitis
- Polyarteritis nodosa
- Systemic lupus erythematosus

C. Infections (septicaemia, enteric fever, malaria, infectious hepatitis, sub-acute bacterial endocarditis)

D. Remote effects of extracranial neoplasms

E. Blood dyscrasias

F. Physical factors (heat stroke, hypothermia, radiation damage)

3. Intoxication

A. Drugs (anticholinergics, antihistamines, antiparkinsonians, antidepressants, anticonvulsants, antiarrhythmics, antihypertensives, antibiotics, antineoplastics)

B. Alcohol

C. Industrial poisons

4. Withdrawal from Drugs & Alcohol

Victims of Trauma

Patients are brought to the emergency department as a consequence of physical or sexual assault. Most of these are women who have suffered trauma either due to domestic violence or are victims of rape. In addition to attending to the physical injuries suffered by such patients, physicians should pay particular attention to the accompanying psychological distress. The manner in which the initial interview is conducted is important. While examining female victims of sexual assault, physicians should exercise considerable tact and sensitivity. In such cases it is preferable that a woman doctor conducts the initial examination and interview. Patients should be helped and encouraged to talk about the incident in detail. This has a cathartic effect and may prevent the later development of a potentially more serious illness: post-traumatic stress disorder. It is also important to document carefully, patient's verbatim account of the assault and the various injuries suffered by the patient, as they may be required later for police investigation. Such patients have to be advised about contraception and testing for sexually transmitted diseases, including HIV. Many of these patients need further psychological counselling and need to be referred to appropriate agencies. Female children can be victims of sexual assault and the perpetrator of the crime usually is a family member such as a father, step-father or a sibling.

Children may present to the emergency services with serious physical injuries. Presence of multiple injuries and fractures at various stages of healing should raise the possibility of "battered child syndrome". Often times the offender is a close family member.

Admission to an in-patient unit is generally recommended. It serves the dual purpose of removing further threat of violence and to address the emotional and psychological needs of the child.

Conclusion

The world over, there has been an increase in the number of psychiatric emergency visits to the hospital. Most often initial services are provided by non-psychiatric physicians. In this chapter an attempt has been made to acquaint the non-psychiatric physicians with the presentation, assessment and initial management of common psychiatric emergencies. It is important that physicians in the emergency department should have a working knowledge of various legal issues related to psychiatric emergencies. These are covered under the Mental Health Act (1987), Narcotic Drugs & Psychotropic Substances Act (1985, Amendment, 1988) and various sections of the Indian Penal Code 1860 (Sec. 84, Sec. 335, Sec. 305) . To reiterate, the goals of the emergency mental health services, as exemplified by the "triage model" are, easy access to emergency care, a comprehensive bio-psycho-social assessment and prompt initiation of treatment. It is imperative that general hospital psychiatrists must work in close collaboration with emergency department personnel in providing regular inputs by way of training and to ensure continuity of care to patients seeking help from the emergency services.

Suggested Reading

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The Management of the Severely Injured Patient

Dr. John K. Gosnold

Hull & East Yorkshire Hospitals, United Kingdom

Introduction

Every year, throughout the world, more young people die as a result of trauma than any other disease process. In the USA, for every trauma death, 19 other persons are hospitalised and 354 receive immediate care. The lifetime cost of trauma in 1990 in the USA was estimated at $215 billion.

In Great Britain the figures are not dissimilar although the pattern of injury is different with blunt trauma far exceeding penetrative injury.

A major problem in both countries is that many deaths are avoidable and, with good management, the morbidity outcomes could be considerably improved and the lifetime costs significantly reduced. In 1988 a Royal College of Surgeons Report on the outcome of trauma deaths suggested that a significant number of deaths from trauma were avoidable and that most of the deaths were due to conditions easily managed by GOOD District General Hospitals.

Since 1988 significant improvements have taken place to improve the standard of care afforded to the severely injured patient. Immediately following the Report, which called for the establishment of American style Trauma centres in the United Kingdom, a comparator study showed that there was 'no significant difference' between a Trauma Centre and a GOOD District General Hospital. As a consequence more effort has been put into the improvement of the District General Hospitals and the establishment of Trauma Systems.

ATLS guidelines show that there are 3 phases in the management of the patient suffering from Life threatening injury which must be considered:-

1. The accident itself, at which stage some injuries are sustained the outcome of which is immediate and untreatable.
2. The 'Golden Hour', when nearly all the avoidable deaths occur.

and

3. The period following admission to hospital, when patients die as a result of complications - some of which may be avoidable and due in part to the wrong treatment in the first two stages.

Phase 1. Pre-Hospital Care

This phase of the treatment is being addressed elsewhere in the Conference programme but it is pertinent to suggest that the best way to treat an accident is to avoid it. Accident Prevention methods, therefore, become extremely important in reducing the number of unavoidable deaths.

Injury prevention counter measures can be directed at human and behavioural factors, vectors of energy and environmental factors. These counter measures are best implemented through the "4 E's" of injury prevention:-

1. Education.
2. Enforcement.
3. Engineering.
4. Economic Incentives.

Once an accident has occurred the only person who might be able to prevent immediate avoidable death will be the Bystander who may help by:

1. Calling for help.
2. Administering first aid, - which realistically will only help with simple problems such as an obstructed airway and the correct positioning of the patient.

The competence and co-ordination of the Emergency Services are the factors most likely to save the life of the patient.

A structure must be established which allows, within available resources, for the patient to be extracted (if trapped), given immediate and life saving treatment and then transported rapidly to the nearest hospital with provision for treating a patient with multiple injuries (i.e., a patient with a Trauma Score of >8 or an ISS of >15).

A robust triage system should be in place, which allows for sensible decisions to be taken as to which hospital the patient is to be taken.

Once freed for transportation the patient should be taken as quickly as possible to a Surgical facility. The policy of 'Stay and Play' accepted in the 80's is generally no longer accepted, having been replaced by a policy of 'Cut and Run' and 'Grab and Dash'.

Common sense should however prevail depending on the type of transport being used and the time to the nearest centre.

It must be sensible to obtain venous access before peripheral shutdown occurs or to treat a Tension Pneumothorax before the situation is irretrievable and the chest tamponades.

Otherwise simple first aid methods of ensuring an Oxygen supply to the cerebral tissue will suffice.

The Triage decision used to distribute patients throughout a Trauma Service should be based on:

1. The mechanism of injury
2. The Revised Trauma Score.

or, more simply,

3. The CRAMS System.

Another, and perhaps more important, aspect to having a good process is the speaking of a common language. That language is the language of ATLS.

Although regarded by some as being too didactic ATLS has enabled a standard of care which is understood by all, addresses simple issues with a standard methodology and therefore ensures a commonality of practice. Certainly it has manifestly ensured a team approach understood by the Medical and Nursing Teams and the Emergency Services.

Within the Standard Setting Exercise of a Trauma System the use of the Trauma Score and the Injury Severity Score, provided the collection of the data is detailed and complete, will enable the use of the TRISS methodology and an accurate predictor of outcomes. Audit of the patient sustaining multiple injuries can then be carried out and the systems altered accordingly.

Phase 2. In the Emergency Room

The management of the patient in the Emergency Room will almost certainly affect the morbidity outcome of the patient more than the mortality. An Emergency Department, which agrees to accept patients suffering from multiple injuries, should have been accredited to receive Major Trauma and, therefore, have the necessary facilities and organisation.

Advanced planning for the patient's arrival is essential. Communication with the Emergency Services should already have been carried out giving some idea of the patient's condition and the particular Specialist Teams likely to be required in the Receiving Room to support the Emergency Team.

An absolute basic team should include an Emergency Consultant trained in ATLS, a support Emergency doctor (sometimes in training), a team of trained nurse, an Anaesthetist, a Radiologist, and Laboratory personnel.

The equipment necessary should have been organised and prepared with warm Ringers Lactate available and ready to infuse.

On arrival the Emergency Team should carry out a rapid and immediate Triage, whilst simultaneously carrying out any resuscitation procedure necessary.

Treatment is sequential and priortised:

1. Airway, with protection of the Cervical Spine.
2. Breathing/Ventilation.
3. Circulation with immediate Haemorrhage control, both external and internal.

The Primary Survey, during the Triage process, will enable a rapid decision as to which Specialist Teams are required to support the Emergency Team, and then

to make a priority Care Pathway following the admission of the patient to hospital.

The overriding priority must always be to ensure an oxygenated supply of blood to the brain, thus minimising secondary cerebral damage (Table 1).

If it is possible to stabilise the patient then a more sedate and progressive treatment process can be carried out.

If not then a more urgent and aggressive approach has to be taken to deal with:

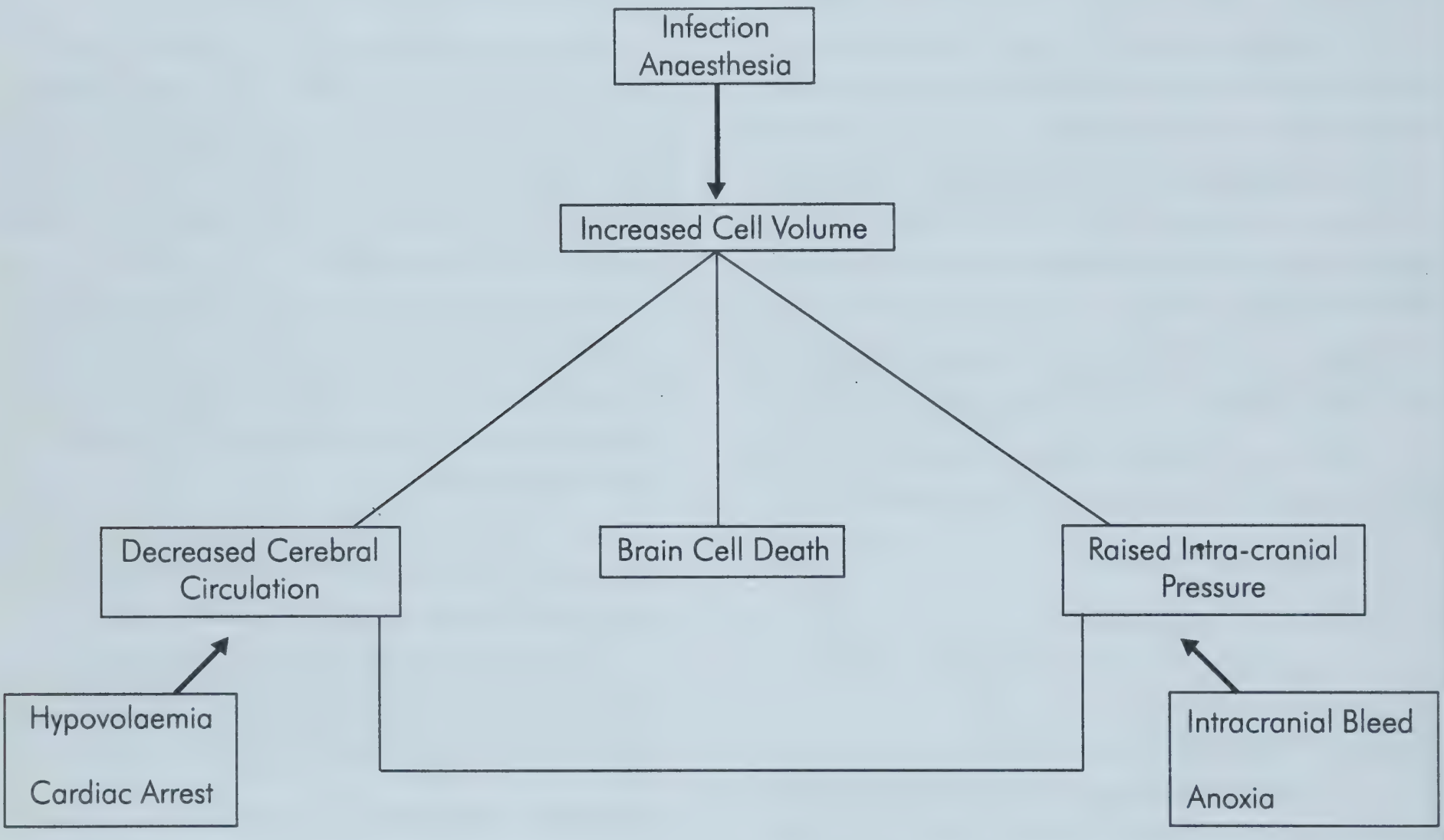
i) The Tension Pneumothorax - immediate thoracocentesis with either a wide bore needle in the second intercostal space in the mid-clavicular line, or a chest drain, both before X-ray and prior to intubation and positive pressure ventilation.

ii) Any physical disruption of the airway requiring a surgical airway, either tracheostomy or, if possible, a crico-thyroidotomy.

iii) Internal bleeding - transfer to theatre and operate.

iv) Intracranial haemorrhage - do CT Scan and transfer to theatre if necessary, whilst, at the same time, remembering to preserve other systems as much as possible.

Table 1. Secondary Cerebral Damage



A **Team Approach** is essential following the accepted sequence of:-

Airway

Breathing

Circulation

Disability - Neurology

Exposure and Environment. - undress and keep warm.

The resuscitation room should therefore be equipped with the tools to allow for:

1. Immediate assessment

Pulse, Blood Pressure, Oxygen saturation, Core temperature, Ventilation rate, Arterial blood gas monitoring.

2. Airway

Oral/nasal airway.

Endotracheal Intubation

Surgical airway.

Cricothyroidotomy

Tracheostomy

Thoracocentesis

3. Breathing/ventilation

Bag & mask.

4. Circulation

Intravenous access 2 large calibre intravenous catheter.

Fluids and Giving sets.

Fluid warmer and pump.

Central lines.

Cut down sets.

5. Disability

X-ray - Chest, cervical spine, pelvis.

C.T. scan.

6. Exposure

Big scissors.

Collars, spinal boards, etc.

Fluid warmer.

Cardiothoracic Bypass Team!

7. Monitor adjunct

E.C.G. Monitor.

Urinary catheter and bag.

Gastric catheter.

Only when the patient is stable and under control should you go onto Secondary Survey, relating this to the mechanism of injury. A Polaroid photograph of the scene is often useful.

The history should have been obtained from the Ambulance crew who should have stayed in the Resuscitation Room to assist and to learn. They are part of the team.

The patient can then be transferred out of the Resuscitation Room, a planned care pathway having been agreed with the Physicians who are going to be responsible for the ongoing treatment process.

The most important thing at this stage is that one **named** person is responsible for taking over the control of the patient when they leave the Emergency Department.

Summary

The treatment of a patient with multiple injuries starts even before the accident with proper accident prevention planning.

Following the accident, and after Bystander First Aid, a co-ordinated team approach with personnel who are used to working together and who use the same protocols will obtain the best outcome.

The vital thing is to have an organised Trauma System.

Early Care of Cervical Spine Injury

Dr. Lester D'Souza
Associate Professor, Department of Orthopaedics,
Fr. Muller's Medical College, Mangalore

A 1989 report estimated that about 6,000 occupants of passenger cars die annually as a result of broken neck and the further 600 people become quadriplegics. Vehicle crashes cause about 50% of the spinal cord injuries while falls, sports (water) and penetrating injuries cause the rest. 3 to 25% of spinal cord injuries occurred during courses of transit or early treatment.

Pre Hospital Care

Pre hospital care is provided by E.M.T.A. (emergency medical technician ambulance), E.M.T.P. (P for Paramedic) and nurses. EMTA is trained in basic life support, EMTP to intubate, I.V. therapy and are trained in ACLS. Nurses can insert chest tubes and airways.

Emergency medical care has also witnessed the development of good hospital care in the form of trauma centres, so that local hospitals can be bypassed. Pre hospital care begins at the site of the accident and is divided into evaluation, resuscitation & immobilization, extrication and transport. Evaluation begins by a primary survey to assess A,B,C. As part of this it is also important to evaluate a patient for a possible cervical spine injury. An unconscious patient or one who complains of pain in the neck or tenderness should be carefully examined. A more thorough head to toe secondary survey is then performed to determine to which faculty a patient should be transported. Resuscitation is begun simultaneously. All patients with a suspected cord lesion should be given supplementary oxygen. If a face mask cannot maintain a desired oxygenation an airway may be used. If a patient has a respiratory arrest, an intubation will be required. If intubation is not possible, an obturator airway should be used. Circulatory support is provided by I.V. fluids and external bleeding should be also controlled.

The helmet should be removed carefully without moving cervical spine. The neck is then immobilized in a Philadelphia hard collar and the patient secured to a long board with straps and sand bag placed on either side of the neck. Patient is then transported safely with minimum hazard and minimum time to the trauma center.

Hospital Care

The hospital care consists of 1. Primary Survey, 2. Resuscitation of life threatening injuries, 3. Secondary Survey, 4. Definitive Care, 5. Stabilization and transport. The treatment is performed by the trauma team. To achieve maximum patient survival one must adhere to the principle that initial attention is directed solely at recognizing and treating life threatening conditions. This is possible best by an accomplished trauma team that sets priorities to treatment. The team should be available before the patients arrival to the trauma center through telephonic contact from the scene of the injury.

During the primary survey, life threatening conditions are identified and management is begun simultaneously. This is performed in the following sequence.

A: Airway maintenance with cervical spine control

B: Breathing

C: Circulation with hemorrhage control

D: Disability

E: Exposure

Table A: The AVPU Method for Evaluating Level of Consciousness

A - Awake and alert
V - Responds to verbal stimuli
P - Responds to painful stimuli only
U - Unresponsive to external stimuli

When immediately life-threatening injuries have been found (if present) and treated, and resuscitation has begun, the third phase of the initial evaluation, the secondary survey, is begun. This phase involves a complete, head-to-toe physical examination of the patient. At this point, a more thorough evaluation of the patient's neurologic status should be made. This should include use of reliable, objective scale for assessing the patient such as the Glasgow Coma Scale. The goal of this phase is not only to find all injuries present, but to ensure that no new life-threatening injuries have appeared or recurred despite therapy. In concert with this, a patient history should be obtained. Although this should be as complete as possible, the minimal amount of information necessary to care for the patient is contained in an "AMPLE" medical history, as shown in Table B. The patient's tetanus immunization status should also be ascertained.

Table B: "AMPLE" Medical History

A - Allergies
M - Medications currently being taken by patient
P - Past illnesses
L - Last meal
E - Events preceding the injury

The fourth phase of the initial assessment follows and is termed "definitive care". Here the appropriate specialists and therapies for the patient's injuries are marshalled and treatment is begun. This phase is followed by the fifth and final stage, stabilization and transportation, as the patient is readied for transfer from the ED to the next level of intensive care unit, or ward, or an interhospital transfer to a regional trauma centre.

Although the risk of a cervical spine injury being present is acknowledged in the primary survey with the admonition that the airway must be made patent while control of the spine is maintained, the actual evaluation for cervical spine injury belongs in the secondary survey. During the secondary survey, as the neck is examined and neurologic examination is performed, important historic details should be obtained from the patient, paramedical personnel, police, or witnesses to the accident. The mechanism of injury must be elicited in detail. For example, for the victim of a motor vehicle accident, the ED physician should ascertain whether or not the accident was multivehicular, and if so, the angle(s) of incidence, speed of the vehicle(s), deformities of the car, steering wheel, windshield, or dashboard; whether the patient was the driver or a passenger; where the patient was seated in the car; whether seatbelts were worn; whether the patient was ejected from the car; etc. A history of neck or neurologic injury should also be obtained. Symptoms of cervical spine injury, as shown in Table C include a history of loss of consciousness, neck pain or stiffness, weakness, or paraesthesia. Signs pointing to a cervical spine injury include unconsciousness, head or maxillofacial injuries, paralysis or paresis, paraesthesia, hematoma, crepitance, or abnormal bony contour, and wounds to the neck with penetrating trauma. A trauma patient who is unconscious or has an altered mental status, even if caused by alcohol or drugs, should be presumed to have a cervical spine

injury and have the cervical spine protected until the physical and radiologic evaluation ensure injury is not present. The ED physician should, likewise, be suspicious that a patient with injuries from the level of the clavicles cranially may have a cervical spine injury as well. Finally, the physician must be aware that in some multiple-trauma patients, the pain from other injuries such as a femur fracture or clouding of the sensorium by alcohol or other drugs, may obscure the symptoms of a cervical spine injury without neurologic deficit. If the unwary physician does not question the patient carefully about neck complaints, no matter how slight, and protect the spine until certain that an injury is not present, a cervical spine injury may be missed with potentially disastrous results for the patient. Most of the reports of painless cervical spine fractures in the literature may, perhaps, be explained by this mechanism.

Table C: Symptoms and Signs Suggestive of Cervical Spine Injury

Symptoms:
History of unconsciousness
History of fall of more than three times patient's height
History of high-speed motor vehicle accident
Neck pain or stiffness weakness
Paraesthesia
Signs:
Unconsciousness
Head injury
Maxillofacial injury
Paralysis or paresis
Parasthesia
Cervical hematoma or ecchymosis
Cervical crepitance of abnormal bony contour
Cervical tenderness
Penetrating cervical trauma
Altered mental status caused by drugs or alcohol

Part of the evaluation of all multiple-trauma victims is a complete neurologic examination.

One of the most important distinctions the initial examiner can make, at least for patient prognosis, is whether the spinal cord lesion is complete or incomplete. This classification determines whether there is total loss of sensory, motor, reflex, and sympathetic activity below the presumed level of injury, or if a portion of any of these functions persist. Depending on the pattern of lost and retained activity, several spinal cord syndromes can be described. Prognostically, findings indicating an incomplete spinal lesion are important because the chance of useful functional recovery is more likely in such cases. To diagnose an incomplete lesion, the attending physician must give special attention to the genital reflexes and the phenomenon of sacral sparing. The genital reflexes include the anal wink, bulbucavernous reflex, and penile erection (priaprism). The anal wink is elicited when gentle stroking of the skin in the perianal region produces contraction of the anus. Loss of this reflex and loss of voluntary contraction of the anal sphincter during rectal examination, coupled with the absence of motor control below the upper extremities, indicate complete motor paralysis is present. The bulbocavernosis reflex is stimulated by either a pull on the urethral catheter, which stimulates the trigone of the bladder, or by a gentle pinch of the glans penis, resulting in a reflex contraction of the anal sphincter around the examiner's finger. This reflex is important because if the patient is not in "spinal shock" (discussed below) and there is no sensory or motor sparing, but this reflex is present, then a complete spinal cord lesion is present. Sacral sparing is defined as the presence of normal perianal sensation. Again, finding sacral sparing or that the patient retains any ability to discriminate between a sharp and dull stimulus, indicates an incomplete spinal cord lesion. With either of these findings, the prognosis for eventual recovery is improved.

Having decided, based upon the history and physical examination, that a cervical spine injury may be present, the examining physician must obtain the appropriate radiologic studies. Much controversy exists over how many and what reoentgenograms should be obtained. Currently, the debate centers on how extensive the basic cervical spine x-ray series should be. On one hand, a technically adequate cross-table lateral cervical spine reoentgenogram should allow a diagnosis or suspicion of significant injury in upto 90% of patients. On the other hand, some have advocated routine use of computed tomography (CT) scan for all suspected spine injuries. No one test, or even series of tests, is likely to achieve 100% specificity and sensitivity in this area. Also, management of other life-threatening injuries may necessitate a less than "full" cervical spine series. For example, the patient who is, haemodynamically unstable and requires expeditious transfer to the operating room cannot be delayed in the Radiology room while multiple views of the cervical spine are obtained.

In such a case it may be sufficient to obtain a technically adequate, portable, cross-table lateral cervical spine roentgenogram and then proceed to surgery with the patient's cervical spine still protected. If this view is completely normal, the anesthesiologist, for example, may be able to intubate the patient's airway with less trepidation. If any abnormalities are seen in the film, the anesthesiologist may need to modify the techniques and control the airway with nasotracheal intubation, or have a cricothyroidotomy or tracheostomy performed by the surgeon.

Assuming the patient is clinically stable, however, the debate over the basic series involves only whether three or five views of the cervical spine, if normal, are sufficient to "clear" the cervical spine of injury. The most important view is the cross-table lateral roentgenogram of the neck, which should demonstrate most, but not all, unstable cervical spine injuries. An example of a cross-table lateral x-ray cleared by the radiologist, but later found to have a significant dislocation are common. The open-mouth odontoid view is necessary to provide further details on the cervicocranium, whereas the anterior-posterior view provides added details on the lower cervical vertebrae. Although all agree these three views are mandatory in a "complete" cervical spine x-ray survey, a number of physicians, radiologists in particular, feel that two oblique views of the cervical spine are also necessary. We are of the opinion that the three-view cervical spine series is the minimum that should be obtained by the physician caring for all patients who are victims of multiple trauma.

Special studies that may be obtained include the oblique views mentioned previously, and if the lower cervical vertebrae cannot be visualized down to their articulation with the first thoracic vertebrae, a swimmer's view should be obtained. Similarly, if suspicion exists that a fracture may involve the articular pillar, then a pillar view should be ordered. Flexion-extension views in the lateral projection may be ordered, after appropriate consultation and under physician supervision, if a ligamentous injury resulting in an unstable cervical spine is suspected. Finally, the orthopedic or neurosurgery consultant may need further information about the location and extent of a cervical spine injury and obtain tomograms, CT scans, or three-dimensional CT views.

TREATMENT

A. Immobilization

Pre hospital care personnel usually immobilize patients before their transport to the emergency department. Any patient with a suspected spine injury must be immobilized above and below the suspected

injury site until injury has been ruled out by roentgenograms.

Immobilization with a semirigid collar does not necessarily assure stabilization of the cervical spine. Immobilization to a spine board with sandbags may be more effective in limiting certain neck motions. Cervical spine injury requires continuous immobilization of the entire patient with a semirigid cervical collar and cardboard before and during transfer to a definitive-care facility.

B. Intravenous Fluids

Intravenous fluids usually are limited to maintenance levels unless specifically needed for the management of shock. Hypovolemic shock can usually be differentiated from neurogenic shock by tachycardia in the former and bradycardia in the latter. If in doubt, elevate the legs, and if the blood pressure does not improve, administer a fluid challenge. Overzealous fluid administration can easily cause pulmonary edema in a spinal cord injury patient. A urinary catheter is inserted to monitor urinary output and prevent bladder distention.

C. Medications

Proper limitation of fluid intake usually obviates the need for diuretics. The value of steroids is controversial, however, they are frequently used in the early management of spinal injuries. Steroids used should be determined in consultation with a neurosurgeon. Ideally, a management protocol is established by consultation.

D. Transfer

Patients with unstable fractures or a documented neurological deficit should be transferred to a definitive-care facility. For physicians who do not routinely see such injuries, the safest procedure is to transfer the patient after phone consultation with a specialist. Avoid unnecessary delay. The patient should be stabilized; necessary splints, backboards, and/or semirigid cervical collar applied; and the patient transferred under medical supervision. Remember, high cervical spine injuries can result in partial or total loss of respiratory function. These patients are best managed by maintaining an adequate airway and ventilation. If endotracheal intubation is indicated, the nasal route should be used if possible.

Approach to a Patient with Fracture Pelvis in the Emergency Department

Dr. Rajagopalan
Professor and Head, Department of Orthopedics,
St. John's Medical College, Bangalore

Fractures of the pelvic ring have long been recognized as a source of major morbidity and mortality. Reported mortality from fractures of the pelvis range from 5% to 20%. Overall mortality in a series of 604 fractures of the pelvis reported by Rothenberger was 12%. Most deaths are secondary to massive hemorrhage and associated injuries initially and to sepsis and multi-organ failure later.

Although some fractures are caused by minor falls, particularly in the elderly, most are the result of high energy trauma.

Classification

Clinical literature before the 1950s reflects the dilemma, indecision and lack of consensus regarding the definitive approach to classification, diagnosis and management of fractures of the pelvis

The most commonly followed classification is by *Tile et al.*

Tile's classification of pelvic disruption:

Type A

Stable – A1- fractures of the pelvis not involving the ring

A2- stable, minimally displaced fractures of the ring.

Type B

Rotationally unstable, vertically stable

B1 – open book

B2 – lateral compression, ipsilateral

B3 – lateral compression, contralateral (bucket handle)

Type C

Rotationally and vertically unstable

C1 - Rotationally and vertically unstable

C2 – bilateral

C3 – associated with acetabular fracture

Investigations

- **Pertinent Radiographs:**
  - *AP view*
  - *Inlet* and *outlet* views:
  - *Judet views*
- **Push-Pull Views:**
  - used to evaluate dynamic displacement of the pelvis;
  - these views are obtained as an *outlet* view with the examiner pushing on the femur for one view and pulling on it for the other;
- **Sacroiliac (SI) Views:**
  - used to visualize the *sacroiliac* joints;
  - the patient is positioned as for Judet views with the central beam directed towards *the sacroiliac joint;*
  - only a CT scan of the pelvis can give a well detailed image of the *SI joint;*
  - neither the iliac or obturator oblique radiographs show this as well as a CT scan.

➢ Lateral View:

- required if *sacral* fracture is suspected;
- the technique is identical to that used for lateral view of the lumbar spine, except that it is centered on the sacrum.

➢ Arteriography:

- arteriography is probably indicated for patients with concomitant pelvic fracture and hemodynamic instability,especially when there is fracture displacement through the greater sciatic notch.

➢ CT Scan:

- CT scanning has become an alternative to peritoneal lavage for the diagnosis of intra-abdominal hemorrhage;

Complications

- Acute Complications
- Complications of Surgical Treatment
- Late Complications

Acute Complications

1. Haemorrhage
2. Associated injuries
   - impotence: occurrs in 37 % of diastasis patients as the result of injury to the nervi erigentes or interruption of the penile blood supply.
3. Abdominal and thoracic Injuries
4. Genitourinary injuries -
5. Neurological injuries
6. Thromboembolic Phenomena.

Complications of Surgical Treatment

1. Complications of surgical exposure
2. Infection

Late Complications

1. Nonunion
2. Malunion

Haemorrhage

Haemorrhage is the most perilous complication associated with fracture pelvis. It may be from the fracture site, a venous tear, arterial injuries, or all three.

- Magnitude of Bleeding:

- the magnitude of blood loss often goes unrecognized.
- the retroperitoneal space may accumulate upto 4 litres of blood before venous tamponade occurs.
- a 3-cm pubic symphysis diastasis will cause doubling of the normal volume of the pelvis, which would allow several units of additional blood to accumulate before a tamponade effect occurs.
- with posterior instability, the average replacement requirement is > 15 units packed cells compared to an average of 5 units packed cells required for pelvic fractures without posterior instability;
- always consider the need for FFP(Fresh frozen plasma) in addition to units packed cells in cases of massive blood loss.

- Associated Injuries:

- as many as 40% of patients who have pelvic fractures have been noted to have an intra-abdominal source of bleeding that contributes to mortality and morbidity after blunt trauma.

- Arterial Bleeding:

- massive blood loss usually occurs from injury to the superior gluteal artery or anterior branches of the internal iliac artery.

- superior gluteal artery injury occurs because of its relation to the SI joint.
- internal pudendal artery injury occurs due to the sharp fascia of the piriformis muscle.
- less often injury to the lateral sacral artery occurs in disruptions of the posterior portion of the pelvic ring.

- arterial injuries are uncommon and are usually seen with open pelvic fractures.
- arterial injuries occur most often in Unstable pelvic fractures -Tile's Type II & Type III;
- arterial bleeding is amenable to a variety of embolization techniques

Methods of Management of Haemorrhage

1. Laparotomy and direct vascular ligation. Useful in penetrating injuries, but not in blunt trauma.
2. Laparotomy and clot evacuation and extraperitoneal packing and internal fixation
3. Application of Pneumatic Anti-shock Garments. Useful for transport.
4. Angiography and embolisation.
5. Open reduction and internal fixation
6. Closed reduction and percutaneous fixation
7. External fixation: advantages
   - prevents gross motion and redisplacement of clots
   - stabilizes the true pelvis
   - decreases bleeding at fracture site.
   - Promotes early patient mobility

MANAGEMENT OF PELVIC FRACTURE

Non Operative Treatment:

should be considered for anterior diastasis of less than 2.5 cm.

Fixation Methods:

1. External Fixation:
   - indicated for SI diastasis or pubic ramus fractures which cannot be treated by open reduction or screw fixation.
   - such as may occur with suprapubic cystostomy or open fractures.
2. Open Reduction and Plate Fixation:
   - anterior plates: indicated for pubic diastasis greater than 2.5 cm.
   - Retrograde Medullary Superior Ramus Screw: indicated for superior ramus fractures which occur along with an ustable posterior injury.

Cerebral Protection in Head Injuries

Dr. G.S. Umamaheswara Rao M.D.
Professor of Neuroanaesthesia, National Institute of Mental Health and Neurosciences, Bangalore 560 029

Cerebral protection refers to prevention of neuronal death following an ischemic injury. An injured brain is characterised by three groups of neurons: 1. A group of neurons which are necrosed and beyond any possible functional recovery, 2. A group of "threatened neurons," which are subjected to ischemia but have a potential for recovery with appropriate protective strategy and 3. A group of normal neurons. No therapeutic intervention is likely to change the course of the first group and the third group does not require any specific attention. It is the ability of any therapy to ameliorate ischemia in the second group that determines its efficiency in improving the clinical outcome.

Pathophysiologic Considerations

Following head trauma, the primary injury may vary from a minor concussion to severe diffuse axonal injury with brainstem signs. The secondary pathophysiologic events that follow the primary injury result in continued neuronal loss. The intensity and diversity of the secondary events has a major bearing on the outcome of the traumatic brain injury. The fact that the majority of head injury victims present to the hospital in a reasonably preserved neurological status, but a significant proportion of them die later on attests to the fact that there is a potential for preserving the neuronal function, if only the secondary events can be prevented or arrested.

The salient pathophysiologic events with relevance to cerebral protection in head injury patient are as follows:

Cerebral metabolism

Cerebral metabolic rate of oxygen ($CMRO_2$) is depressed following head trauma. The degree of cerebral metabolic depression is correlated with the depth of coma. A linear correlation has been reported between Glasgow Coma Score (GCS) and $CMRO_2$ (1). In patients with severe head injury $CMRO_2$ is generally less than 1.5 ml/100 G/min as against a normal value of 3.0 ml/100 G/min (2). Head trauma may also affect the cerebral blood flow-metabolism coupling to a variable extent. An intact CBF-CMR coupling results in a lower CBF value in a patient with head trauma. A normal or higher-than-normal flow in the presence of depressed metabolism represents hyperaemia. Obrist et al showed that a low flow state representing a normal CBF-CMR relation occurs in 45% of head injured patients. Absolute or relative hyperaemia representing impaired CBF-CMR relation is seen in 55% of head-injured patients (1).

Cerebral blood flow

Both ischemic and hyperaemic ranges of cerebral blood flow have been documented following head injury. In general, ischemic levels of CBF are seen very early following the head trauma (6-12 hours) and are responsible for early mortality. Hyperaemia is a relatively late phenomenon peaking at around 24-48 hour. While ischemia leads to cellular death directly, hyperaemia raises the intracranial pressure and leads to herniation and brainstem ischemia.

CBF changes following head trauma have been divided into 4 phases (3): The first phase lasting up to 12 hours is characterised by very low blood flows, following which blood flow increases rapidly to reach hyperaemic level within 24 hours. The hyperaemic phase lasts up to about 72 hours and is followed by a stage of progressively decreasing CBF lasting for about

20 days. This delayed ischemic phase is followed by a phase of recovery of CBF to normal values. The degree of hypoperfusion during the delayed ischemic phase is an important determinant of the delayed mortality.

Autoregulation

A variable degree of impairment of autoregulatory control of cerebral blood flow follows head trauma. The lower limit of autoregulation - normally believed to be 50-60 mmHg MAP - is elevated following severe head injury. So a higher cerebral perfusion pressure needs to be maintained to protect the brain from ischemic insults resulting from systemic hypotension. The time course of autoregulatory impairment in head injury is not clearly understood. But most studies seem to agree that it is preserved, at least partially during the first 24 hours (2).

Vascular response to carbon dioxide - which ensures a linear relationship between $PaCO_2$ and CBF within physiological ranges of $PaCO_2$ - is preserved till late in the course of head trauma. Loss of response to CO_2 carries a poor prognosis.

Raised Intracranial pressure

Raised ICP has been documented in 50-70% of all severe head injuries (4). Uncontrolled intracranial hypertension leads to cerebral ischemia by decreasing cerebral perfusion pressure (CPP) and is responsible for 58% of mortality in head injuries. Many studies have shown improvement in outcome with aggressive management of raised ICP. Mass lesions such as haematomas, cerebral hyperaemia, brain oedema are responsible for raised ICP.

Cellular Events

Ischemia unleashes a host of biochemical events, which culminate in the death of neurons. Some of the important cascades involved in the process of cell necrosis are mediated by calcium, excitatory amino acids (aspartate and glutamate), free-radicals (superoxide and hydroxyl ions), lactacidosis, ionic fluxes related to potassium and magnesium and inflammatory mediators such as cytokines.

Massive sympathetic discharge is known to occur following head trauma. The concentration of endogenous catecholamines is inversely related to the Glasgow Coma Score of the patient. The high sympathetic outflow may have an impact on cardiovascular function resulting in hypertension, tachycardia, myocardial ischemia and neurogenic pulmonary oedema.

Extracranial Sequelae

A number of extracranial sequelae mitigate against the recovery of an injured brain. Important among them are: hypo-/ hypertension, hypoxia, hyper- /hypocapnia, respiratory irregularities, neurogenic pulmonary oedema, electrolyte imbalances, hyperglycemia, hyperthermia and systemic infections.

Therapeutic Strategies

The foregoing discussion on diverse pathophysiogic pathways suggests that no single therapeutic intervention is likely to be uniformly successful. A multi-pronged approach addressing the individual events is probably more rational. Various clinical trials utilising different pharmacological and non-pharmacological interventions with established pathophysiologic basis have yielded suboptimal results. As a consequence, as of now, the main thrust in protecting an injured brain seems to lie on careful prevention of systemic events that are likely to complicate the primary injury. The following discussion highlights the role of extracranial management and specific treatment strategies in protecting the injured brain.

Management of Extracranial Organ Function

Adequate management of extracranial organ function is essential to prevent secondary neuronal loss.

Haemodynamic Management

The aim of haemodynamic management is to maintain euvolemia and adequate mean arterial pressure to achieve the required cerebral perfusion pressure. Central venous pressure is ideally maintained around 5-10 mmHg and the pulmonary capillary wedge pressure between 10 to 14 mmHg. Supranormal values of CVP or PCWP may not improve the cerebral perfusion any further, but may contribute to the formation of cerebral oedema. When the CVP is less than 5 mmHg, additional fluids (cystalloids, colloids or blood) should be given. If the CPP is inadequate despite a CVP of 10 mmHg or a PCWP of 14 mmHg, inotropic or vasopressor support is indicated. In young previously healthy adult patients norepinephrine in a dose of 0.01-0.20 ug/kg/min is a good choice. If the urine output falls or the patient develops metabolic acidosis, renal dose of dopamine should be started. Dopamine is an ideal inotrope in older patients. Dobutamine may be added to norepinephrine if the cardiac index falls to less than 3 $L/min/m^2$.

Respiratory management

The injured brain is highly vulnerable to hypoxia. Both hyper- and hypocapnia are detrimental to the injured neurons. Therefore, the respiratory management should aim to achieve a PaO_2 of at least 90 mmHg and near-normocapnia (30-35 mmHg of $PaCO_2$). This warrants sedation/muscle paralysis and artificial ventilation. Respiratory infection is very frequent in these patients. Fever leukocytosis and x-ray infiltrates suggest pulmonary infection, which should be treated with appropriate antibiotics based on culture reports.

Fluid management

It is essential to avoid excessive dehydration. Maintenance requirement should be calculated at a rate of 1.5-2.0 ml/kg/hour. Excessive urinary losses due to diuretics have to be replaced. If the serum sodium is more than 140 meq/L, half-normal saline is an ideal fluid for maintenance therapy; if it is less than 140 meq/L isotonic saline is indicated. The losses of potassium due to diuretic therapy need adequate replacement. Specific syndromes of fluid and electrolyte imbalance (diabetes insipidus, SIADH etc.) should be recognised and treated early.

Temperature regulation

Normothermia should be maintained. Fever should be controlled by appropriate treatment of infections. Symptomatically, hyperthermia may be controlled by paracetamol, cold saline infusion and cooling blankets.

Control of infections

The major sources of infection in unconscious head-injured patients are chest, urinary tract, operative wounds, septicemia and meningitis. Relevant microbiological samples should be sent for culture and antibiotic therapy based on the sensitivity reports.

Specific Strategies

Management of Intracranial Hypertension

Improved outcomes have been reported when ICP was maintained below 15-20 mmHg. Most authors treat intracranial hypertension when the ICP exceeds 20 mmHg. Contusions in temporal fossa or deep frontal region need to be treated even at 15 mmHg as they may cause herniation even at a lower ICP level.

General measures

Normovolemia, satisfactory oxygenation and ventilation should be ensured. Fever should be avoided as each degree centigrade of fever increases ICP by several mmHg and also increases the metabolism. Elevation of head by 15-30° not only decreases ICP but also avoids ICP spikes by allowing cerebral venous outflow. Normovolemia should be ensured before

head-elevation as hypotension occurring as a result of head-elevation is likely to decrease CPP. Head should be maintained in a neutral position such that there is no pressure on neck veins. The role of anti-seizure prophylaxis is unclear. But in a patient with intracranial hypertension, seizure activity may increase ICP precipitously. In a patient under pharmacologic paralysis, the only indications of a seizure are tachycardia, increased ICP and fluctuation of blood pressure. Phenytoin is the most commonly used drug for the seizure prophylaxis; sodium valproate is another alternative.

Hyperventilation

Since vascular reactivity to carbon dioxide is preserved till late in a head injury, hyperventilation is very effective in decreasing the CBF, CBV and ICP. Hyperventilation may avert impending herniation. But it may also produce cerebral ischemia. Obrist et al demonstrated that 45% of the severely head injured patients exhibited hypoperfusion and hyperventilation increased the arteriovenous oxygen difference in these patients (1). Gopinath etal, in a series of 116 patients monitored by jugular venous oxygen saturation, could attribute 27% of episodes of cerebral oxygen desaturation to a $PaCO_2$ less than 28 mmHg (5). Studies by Rosner et al (6) and Marion (7), where $PaCO_2$ was maintained at 35 mmHg, have confirmed that hyperventilation is not necessary in the management of severe head injury. Hyperventilation is probably most dangerous in the first 24 hours when the CBF is normally very low. If hyperventilation becomes necessary to control ICP, tissue oxygen delivery may be optimised by increasing the FiO_2 and maintaining the haematocrit at 30%.

Diuretics

Osmotic and loop diuretics help to decrease brain water content and ICP. Mannitol, the most commonly used osmotic diuretic, does not cross the intact BBB and decreases extracellular brain water content both in normal and abnormal areas of the brain. In addition, it also decreases the viscosity of blood and induces a reflex vasoconstriction. Frusemide, a loop diuretic, is also used to decrease brain water content. Frusemide induces systemic diuresis as well as decreases CSF production. It is used alone in a dose of 0.5-1.0 mg/kg or in a dose of 0.15-0.30 mg/kg in combination with mannitol. The combination has a synergistic action and prolongs the duration of action of mannitol. But it may lead to more profound diuresis and more severe electrolyte disturbances.

Barbiturates

When conventional measures fail to control intracranial hypertension, barbiturates may be used as a second line of therapy. Substantial evidence exists to show that barbiturates are effective in lowering ICP. But outcome improvement is limited to only a select group of patients with refractory intracranial hypertension (8). Prophylactic administration does not seem to have any role in improving the outcome.

Some of the potential mechanisms through which barbiturates protect the brain are: i) Sedative effect leading to cerebral metabolic depression; $CMRO_2$ can be decreased by about 40% with a dose of barbiturate achieving electrocerebral silence, ii) cerebral vasoconstriction and a reduction of CBF and CBV; the lowering of CBF is effected by flow-metabolism coupling, iii) free-radical scavenging.

Systemic hypotension is the most common complication of high-dose barbiturate therapy. So previous evidence of cardiovascular compromise contraindicates the usage of barbiturate coma. Normovolemia, normal blood pressure, normal cardiac output and urine output should be ensured before commencing the therapy. Patients with high

frequency EEG activity are likely to benefit more than those with low frequency activity. Dopamine, noradrenaline and phenylephrine should be available at hand to treat hypotension resulting from myocardial depression and peripheral vasodilatation. Though the goal of the therapy is to achieve a control of ICP, doses higher than these may be required if it is aimed to achieve an isoelectric EEG. Monitoring the function of CNS during barbiturate coma is limited to ICP monitoring and examination of pupils. Pupillary signs also may be misleading with myosis occurring at low to moderate doses and mydriasis at high doses. Barbiturate therapy should be continued for 24-48 hours after the ICP control is achieved. The therapy should be withdrawn slowly. Abrupt withdrawal may cause rebound intracranial hypertension.

Steroids

Corticosteroids have no role in ICP control in head-injured patients. A recent analysis of 13 randomised controlled studies published till 1996 comprising of 2073 participants shows that there remains a considerable uncertainty over the effect of corticosteroids and large-sample prospective studies are worthwhile (9). This conclusion was later criticised on the grounds that the possible beneficial effect, even if present, is marginal to justify a large randomised study.

On the other hand, 21-aminosteroid compounds (e.g. Tirilazad) are powerful free-radical scavengers and inhibitors of lipid peroxidation. These compounds do not have any glucocorticoid activity. Tirilazad is under clinical trial at present (10).

CSF drainage

CSF drainage is an effective method of ICP reduction if the patient already has an intraventricular catheter. Excessive drainage may cause collapse of ventricles and render further ICP monitoring impossible. Infection and haematoma formation are two major risks of ventriculostomy for drainage of CSF.

Other Pharmacological Agents

Intravenous lignocaine decreases $CMRO_2$ and cerebral glucose consumption. It has some direct cerebral vasoconstrictor effect and blunts ICP response to intubation and surgical stimulus. Though there is some experimental and clinical evidence that it can decrease ICP, its efficacy as compared to other measures has not been established. The only situation where it is definitely useful is endotracheal suctioning. Both intravenous and intratracheal routes have been found to be equally effective.

Etomidate is an effective ICP-reducing agent. The decrease in ICP is brought about through a reduction in CBF, which, in turn, is a result of the reduction of cerebral metabolism. Thus, the mechanism of its action is similar to that of barbiturates. Since mean arterial pressure is maintained better with etomidate than with barbiturates, CPP tends to be maintained well. The major limitation for its routine usage in preference to barbiturates is its association with adrenocortical suppression. It has been suggested that it is possible to reverse this adrenal suppression with supplementation of glucocorticoids.

Propofol decreases $CMRO_2$ and CBF and, thereby, the ICP. ICP has been shown to be decreased both in patients with normal and raised ICP. Since it also causes profound hypotension, the overall effect on CPP and cerebral blood flow - metabolism matching is unclear. Euvolemia needs to be established before using propofol for ICP reduction. Its advantage over barbiturates lies in its short half-life facilitating rapid recovery.

Management of Cerebral Perfusion Pressure

Cerebral perfusion pressure defined as a difference between the mean arterial pressure and the

intracranial pressure is a major determinant of the cerebral blood flow. There are two schools of thought with regard to the optimal level of CPP in traumatic brain injury.

Rosner's Hypothesis

Cerebral ischemia has been recognised as a common and ominous event following head injury. Histologic ischemic brain damage occurs in 90% of fatal head injuries (11). Hypoxia and hypotension are documented in 57% of the patients with severe head injury in Traumatic Coma Data Bank (TCDB) cohort. A single episode of hypotension is associated an 85% increase in the mortality (12). Approximately one third of the patients of severe head injury exhibit a regional or global CBF less than 18 ml/100 G/ min within the first 6-8 h. In addition, nearly one-third to one-half of the patients with severe head injury have some degree of autoregulatory impairment. The most clinically relevant aspect of this impairment is an elevation of the lower limit of autoregulation. Recent studies indicate that a CPP of at least 60-70 mmHg is necessary to optimise CBF in head-injured patients (13). Below this level, the CBF is directly proportional to CPP.

A number of clinical studies documented improved outcome with maintenance of higher cerebral perfusion pressures. In a retrospective study of 180 patients, McGraw demonstrated significantly better outcome when the cerebral perfusion pressure (CPP) was > 80 mmHg than when it was below 80 mm Hg (14). Gopinath et al. (5) showed that mean and lowest recorded CPP correlated significantly with outcome. A CPP of 60 mm Hg was found to be critical below which outcome worsened significantly. When CPP was maintained at or above 70 mmHg and $PaCO_2$ at 35 mmHg Rosner and Rosner reported favourable outcome in 59% and a mortality of 29% (6). With similar criteria for CPP and $PaCO_2$ Marion etal (7) reported favourable outcome in 51% and a mortality of 20%. These results are better than the data of the TCDB with a favourable outcome of 37% and a mortality of 40%.

One of the major concerns in maintaining a high CPP is the increase in CBF and exacerbation of intracranial hypertension associated with it. But recent evidence shows that a very poor correlation exists between high CPP and ICP (15, 6). In fact, if the autoregulation is intact, maintenance of high CPP will help prevent intracranial hypertension. The vasodilatory and vasoconstrictor cascade models proposed by Rosner and Daughton help to understand these relationships. This concept assumes that when autoregulation is intact, low CPP causes cerebral vasodilatation, increases cerebral blood volume (CBV) and ICP. On the other hand, increasing the CPP causes vasoconstriction, decreases CBV and ICP. Although the lower autoregulatory level appears to be in the range of 70 mmHg, the upper limit is less well defined. Experimentally, values in the range of 105 mmHg appear to be well-tolerated for short duration.

The Lund Hypothesis

While the studies by Rosner and Marion stress the need for maintenance of high CPP in the treatment of raised ICP, the Lund group advocates a different approach based on appropriate management of cerebral capillary hydrostatic pressure (16). According to them, following head injury, there is a break-down of Blood-Brain-Barrier (BBB). In these areas with impaired BBB, fluid transfer across the capillary membrane depends on the capillary hydrostatic pressure. With the loss of autoregulation, CBF and capillary hydrostatic pressure become passively dependent on the CPP. This means that the capillary hydrostatic pressure and fluid transfer across the cerebral capillaries passively follow arterial blood pressure changes. The Lund therapy advocates against

high CPP levels as it might exaggerate the brain oedema. They aim to keeping the ICP at a safe level by: 1. decreasing the CBV by causing venous constriction, 2. reducing the extracellular oedema by causing precapillary cerebral vasoconstriction and thereby decreasing the capillary hydrostatic pressure and 3. by normalisation of colloid osmotic pressure. Venous constriction and precapillary arteriolar vasoconstriction is brought about by administration of Dihydroergotamine. Capillary hydrostatic pressure is also brought down by α2-agonist clonidine and β1-antagonist, metaprolol. Both these agents, while decreasing the mean arterial pressure and capillary hydrostatic pressure, do not cause any significant decrease in CBF, as long as normovolema is maintained and the CPP is set above 50 mmHg for adults and 40 mmHg for children. A bolus infusion of 4ug/kg of DHE caused an immediate and significant reduction of ICP by 25% without any effect on CBF and metabolism. The response to DHE was similar irrespective of the cerebrovascular reactivity to hyperventilation (16).

In addition to maintenance of capillary hydrostatic pressure, the Lund concept also stresses the need for the following measures: a) Prompt evacuation of haematomas / contusions which may provoke release of vasoactive substances which could cause vasospasm and break-down of BBB, b) moderate hyperventilation to a $PaCO_2$ of 30-33 mmHg, c) decreasing stress response with liberal usage of benzodiazepines and opioids and also β_1-antagonists in some cases, d) decreasing cerebral metabolism with α_2 agonists or barbiturates, e) Avoidance of hyperthermia and acceptance of mild hypothermia up to 34°C, f) vigorous control of hyperglycemia, g) enteral nutrition and restriction of the usage of parenteral fats and amino acids, h) maintenance of normal colloid osmotic pressure by maintaining serum albumin levels at 4 g/dL to promote resorption of oedema fluid and i) maintenance of haemoglobin level above 12 g/dL. The proponents of the Lund concept report a significant reduction of mortality and morbidity with the new therapy (17, 18).

Mild Hypothermia

Experimental evidence over years has documented the benefits of hypothermia in neuronal preservation. Animal studies have suggested that mild (33°C) rather than moderate (29°C) hypothermia is more optimal to treat cerebral ischemia (19). According to one estimate, 34% of the neurosurgeons advocate 24 hours of hypothermia in the ICU following head injury (20). A survey conducted in 1993-94 indicates that more than 40% of neuroanaes thesiologists are already using mild to moderate hypothermia intraoperatively and 26% of them, in every patient (21). The protection offered by intraischemic mild hypothermia has been attributed to: i) reduction of calcium entry, ii) reduction of glutamate release, iii) reduction of glycine and dopamine release, iv) inhibition of protein kinase C and v) reduction of free radical-triggered lipid peroxidation. The threshold for the protective effect seems to be 36°C (22). It has been shown that one degree of hypothermia (to 36°C) maintains ATP at normoxic levels during a hypoxic insult that depletes ATP by 50% at normothermia (37°C). A 3°C hypothermia more than doubles the preservation of phosphocreatine (23). Marion etal (24), randomly assigned 40 consecutive patients to normothermic (37-38°C) and hypothermic (32°-33°C brain temperature maintained for 24 hour) groups. Hypothermia significantly decreased the ICP (40%) and CBF (26%) during the cooling period and neither parameter showed a rebound on rewarming at the end of 24 hours. At the end of 3 months, there was a trend for better outcome in hypothermic group (12 out of 20 in hypothermic group vs 8 out of 20 in normothermic group). The incidence of systemic complications was

similar between the two groups. The same group, in yet another recent publication, report an improved outcome with mild hypothermia at the end of 12 months in a sub-group of patients with a Glasgow Coma Score (GCS) between 5 and 7 (25). A recent study suggests that mild hypothermia is effective in preventing ICP elevations only in patients without diffuse brain swelling in whom ICP remains higher than 20 mmHg but less than 40 mmHg (26).

Excitatory amino acid antagonism

Cerebral trauma is associated with release of glutamate, which through its effect on NMDA, AMPA and kainate receptors exerts its toxicity by increasing the entry of calcium and sodium into the cell. Increased ECF and CSF concentrations of glutamate, upto several days, have been documented in human traumatic brain injury (27, 28). Randomised controlled trials with the currently available compounds (Eliprodil, Cerestat) have failed to document any major beneficial effects (29). Disadvantageous cardiovascular and behavioral side effects have been documented.

Calcium Channel Blockers

The central role played by calcium in mediating cell damage led to a number of clinical trials utilising nimodipine. In unselected populations, little overall benefit has been conferred (30, 31). Marked trend towards favourable outcome has been documented in patients with traumatic subarachnoid haemorrhage (32, 33).

Free Radical Scavenging and Prevention of Lipid Peroxidation

Free radicals are liberated from mitochondria as a result of activation of the arachidonic acid cascade by high intracellular calcium. These radicals, in turn, cause lipid peroxidation. Polyethylene glycol-conjugated superoxide dismutase is a free radical scavenger that has shown a statistically non-significant increase in favourable outcome (34). Recent work with tirilazad, an aminosteroid compound, which prevents lipid peroxidation, has not shown a significant effect on the outcome.

Magnesium

The findings that intracellular magnesium levels are decreased following traumatic brain injury and magnesium competitively blocks the NMDA receptors, led to investigating its potential as a cerebral protective agent. A preliminary trial of magnesium sulphate in patients with acute middle cerebral artery stroke has provided encouraging result (35). Similar studies are warranted in patients with traumatic brain injury.

In conclusion, despite extensive knowledge on the various pathophysiological mechanisms involved in cerebral ischemia, there has been no therapeutic breakthrough in resuscitation of the injured brain. On possible reason for the failure of any single modality of therapy to significantly influence the outcome in randomised studies is that the pathophysiology is multifactorial and any treatment strategy aimed at one mechanism still leaves the other pathways to continue unabated.

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Disaster Management

Dr. PushpRaj Singh
Department of Emergency Medicine, Christian Medical College & Hospital, Vellore

Introduction

Disasters are unpredictable events, diverse in nature and adversely affect all parts of the globe. The United Nations declared the 1990s the International decade for Natural Disaster Reduction. The International Federation of Red Cross and Red Crescent estimates that by the end of year 2000 the number of people affected annually by disasters of all kinds will be between 300 and 500 million.(1)

Definition

Disaster is a sudden ecological imbalance between man and his environment which causes an unexpected and unusual patient load and needs additional manpower and supplies.

Principles of Disaster Management

"The goal is saving as many victims as possible. In such a situation physicians and surgeons are no long practicing individual-centred medicine but a collective medicine which is governed by specific rules."

A major concern in a disaster situation is to prevent hospitals and resources from being overwhelmed with minor casualties but also provide emergency care to severely affected victims. The solution to this problem is the institution of a medical chain from the area of the disaster to the hospital. At each step in the chain a medical triage of the victims with emergency medical care is performed. The normal hospital organization has to be modified in order to deal with the volume of victims involved.

Plan of Action will be the following Essentials

1. Scene Management

The first medical team at the scene has to evaluate the severity of the disaster. This includes extrication of victims from the affected area. The Disaster Management co-ordinator chooses a place to establish emergency medical camp to provide medical-aid onsite. Such a place must be secure from a disaster evacuation but close to the area. Ideally it should be near a wide road, spacious, dry, warm and well lit, with water, toilets, electricity and telephone facilities. When the siting of the Emergency Medical Camp has been decided, casualty transport begins. If paramedics or doctors are available field care is given. Otherwise first aid workers and stretcher bearers bring casualties to the Emergency Medical Camp.

2. Field Operations

Triage: - (Fr. = sort) It is a system by which patients are classified according to the severity of their injuries. Object of triage is to ensure that "Right patient gets the Right treatment at the Right time". At the entrance of the emergency camp casualties are triaged. Living victims are registered, categorized into absolute emergencies (Priority I & II) and relative emergencies (Priority III, IV & V) by the triage surgeon or physician or nurse, and dispatched either to the Absolute Emergency area or Relative Emergency area. Dead victims are registered, gathered with their belongings and documents that could facilitate further identification, and brought to the mortuary area situated remote from the emergency medical camp.

Clearing Medical Centre

The Clearing Medical Centre is usually a hospital with laboratory, radiology and where elementary emergency surgery can be performed. The goal of the Clearing Medical Centre is stabilizing the condition of victims in order to allow for long transportation times.

After care, victims are placed in a clearing area and evacuated according to priority to near by hospitals for definitive care.

Medical Triage

Triage by Evacuation

This triage is made according to quantity and quality of available means of evacuation, distance to be covered and severity of injuries.

1. **Evacuation is possible, easy and fast:** Triage is based on dispatching victims to specialized centres.
2. **Evacuation is long and difficult:** Care will be given to allow the victims to withstand a long wait.
3. **Evacuation is nearly impossible:** Triage must select those casualties who can be treated on the spot who have a chance of survival.

Triage by Surgical Need

Triage is made according to the severity of injuries.

Classification of Casualties

Carsualties are classified into absolute (Priotity I &II) and relative emergencies (Priority III, IV & V)

Absolute Emergencies: AE

AE needs life support procedures immediately to sustain correct ventilation and haemodynamic parameters. Elementary surgical procedures may be required. Absolute Emergencies are given priority evacuation under the control of a doctor or paramedic.

Priority I

Urgent life support procedures are required to maintain life:

- Ventilatory distress.
- Circulatory distress.
- Extradural haematoma.
4. More than 50% II^0 and III^0 burns.

Priority II

Care is required before transportation, to prevent impending ventilatory or circulatory distress. Casualties are to be transported under medical (or paramedic) supervision. A surgical procedure or intensive care will be needed within 5-6 hours.

1. Thoracic trauma without ventilatory distress
2. Abdominal penetrating trauma without shock
3. Vascular wounds
4. Open long bone fractures
5. Large muscular wounds
6. Cranial trauma with coma
7. Crush syndrome
8. Spine trauma with neurologic signs
9. 30% -50% II and III burns
10. Intoxication with coma
11. Ocular wounds
12. Pulmonary blast injury
13. Drowning with persistent coma
14. 28°C - 32°C hypothermia.

Relative Emergencies: RE

These casualties need immediate nursing care, delayed evacuation without supervision and delayed surgical treatment. There is no vital risk.

Priority III

1. Surgical treatment may be delayed for 18 hours.
2. Closed fractures
3. Open fractures of 'short' bones
4. Wounds of soft tissues and scalp without hemorrhage

5. Cranial trauma with light coma
6. 10-30% II^0 and III^0 burns
7. Shoulder and ankle dislocation without neurologic deficit
8. Articular wounds

Priority IV

Treatment may be delayed beyond 18 hours and seated transportation is possible:

1. Closed limb trauma
2. Small wounds
3. Cranial trauma with improving conscious level
4. Minor abdominal and thoracic contusions
5. Less than 10% burns

Priority V

Victims with extremely benign lesions who, after care, will be able to return home or reach a camp.

Overstepped Emergencies

These victims present with very severe lesions which cannot be immediately treated or have minimal chance of survival. Analgesia is given systemically. The team is informed to avoid useless intensive medical interventions and loss of time.

1. Thoracic and abdominal crush injuries
2. More than 80% II^0 and III^0 burns
3. Arterial bleeding of the neck without possibility of rapid surgical repair. Casualties may be categorized as overstepped Emergencies only in disasters overwhelming medical resources.

Hospital Organisation

Team Co-ordinator

The leader of the team is expected to plan, organise, lead and co-ordinate Disaster management. He is expected to prepare his team in advance so that they are effectively operational, when needed. The Team co-ordinator should NOT be personally involved in the patient care but to supervise the overall activity of all operations.

Planning of Disaster Management

This process begins with analysis of the types of disasters likely in a given area and the vulnerability of the area to those disasters. Epidemiological knowledge of the types of injuries and illnesses caused by disasters is essential to determine appropriate supplies, equipment and personnel needed. Each hospital should prepare a strategic plan to accommodate mass disaster casualties. The Emergency Department is expected to mount an effective emergency response and assistance and play an integral role in strategic policy and planning. Planning for the efficient management of a disaster could include simulation models; "What-if" analysis are performed to predict the consequences of conceivable scenario.(2)

Protocols

Pre-written protocols prove to be of immense benefit, especially when multidisciplinary action is warranted. This process helps to define job responsibilities for the members of the team. Therefore the support of the administration and the heads of the concerned departments is absolutely important in preparing the protocols.

Crisis Staff

Crisis staff is made up of no more than 5 or 6 people including the administrative officer and the more experienced doctor. Crisis staffs are in continuous telephone contact with the persons in charge of the different logistic departments (reception, telephone, telephone exchange, engineering department, dispensary etc.)

Staffing

Personnel involved in Disaster management should be handpicked, briefed, trained and kept on stand by. Periodic refresher courses and mock exercises have proven to be of great use in the activation and effective function of the team. In most disasters, symptoms and injuries follow a pre-set pattern. Therefore, even if the magnitude and the effect may differ, treatment could be standardised. Specialised surgeons and physicians have to adopt themselves to the pathology presented by the victims. Under the direction of the specialists, all medical and surgical staff participates in patient care even if the injuries are outside their specialisation. Accommodation and food needs to be provided for the resting team.

Modification of the Hospital Structure

Ambulance Circuit

In order to avoid a traffic jam at the entrance to the hospital, a circuit for (and only for) ambulances must be instituted with, if possible, a way in and a way out.

Reception Area for Casualties (Triage Desk)

A large, warm, well-lit and equipped space with telephones is used as triage area. This place is not necessarily (and often not) the emergency department. Casualties are registered and dispatched from this area.

Reception Area for Families

To avoid families accumulating in the triage area, a specific place should be provided for them. Administrators inform families the status of their relatives.

Reception for Media

Journalists have to be registered, accredited, gathered in a separate room and updated regularly.

Transformation of Departments

To be able to treat numerous casualties with specific lesions, the designation of some departments has to be changed: after a train accident, medicine departments will receive surgical patients, and after a toxic cloud, surgical departments will receive pulmonary diseases. Stable patients have to be discharged, and elective surgical procedures cancelled.

Stock Control

Pharmaceutical and material reserves of the hospital have to be supplied. Disaster sets prepared beforehand and immediately available allow some autonomy and flexibility.

Strengthening of Specific Areas

Support Services in Disaster

The main players in pre-hospital disaster control are ambulance personnel, fire fighting units, police and bystanders. It has been observed that the members of youth and social service organisations are effective 'front line workers' in disasters especially after some training in disaster management.(3) This phenomenon is observed to be effectively enforced in railway accidents and flood evacuations.

Communications

Telephone exchanges has to be reinforced. Specific lines to headquarters and emergency medical camp must be installed. Establishment of a hot line for relatives' information reduces the volume of communications dispatched by the operator. Discipline in using telephone only for important messages is mandatory.

Reception and Information of Families

To avoid individual or collective maladjusted and aggressive behaviour, great attention must be devoted to the reception and information given to relatives. Social workers are especially competent for this.

Role of Epidemiology in Disaster Management

Disaster Epidemiology scientifically measures and describes the effects of disasters and contributing factors to these effects. Its goals include:

- Assessing the needs of disaster-affected populations
- Efficient matching of resources to needs.
- Further prevention of adverse health effects
- Evaluation of programme effectiveness

Post Disaster Debriefing

The prevalence and protracted course of post-traumatic stress disorder is a reality among disaster relief workers (4). There could be continuing experience of imagery of disaster, which could be incapacitating. Disaster management plans should recognise the psychological impact of natural disaster and the long-term nature of post-traumatic stress disorder and should ensure that preventive mental health programs are implemented.

Conclusion

Disaster management has to be planned. Plans have to be experienced during exercises and improved, re-experienced and re-improved. "In disaster medicine, expect the unexpected"

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Management of Pain in the Emergency Room

Dr. Radhika Dhanpal M.D.

Associate Professor, Dept.of Anesthesiology, St. John's Medical College Hospital, Bangalore

"For all happiness mankind can gain is not in pleasure but in relief from pain".

Pain is an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage. Three-fourth of new patients attending the ER are likely to have some degree of pain. More than half of these patients do not receive an analgesic during the visit because of unfounded beliefs that symptoms may be masked and prevent a definite diagnosis being made or that liberal prescribing may lead to widespread abuse.

Patients vary in their response to pain and the need for analgesia. Some refuse analgesia, others demand it. Personality, ethnic origin, environment, previous experiences all influence the reaction to pain. When choosing the optimum method of pain relief, the patient's age, size, personality, pre-existing medical condition or medication should be considered. The cause, severity and nature of pain are important. Furthermore, plans to admit to hospital or operate may affect the choice of analgesic.

Physiology of Pain - Mechanical, thermal and chemical stimuli activate specialized pain receptors in the periphery. This stimulation is mediated by some of the mediators of inflammation like histamine, leukotrienes, kinins and SRS-A. The impulses are then transmitted by A and C fibers to the dorsal root ganglion. From there they cross the midline and ascend via the lateral and anterior spino-thalamic tracts to the thalamus and other centres of the limbic system.

Goals of Pain Management

- Diagnosis
- Pain Relief

i) History & physical examination if necessary.

ii) Individualized treatment with the help of a pain scale.

iii) Determine patient's level of anxiety and treat it.

iv) Choose the most appropriate analgesic available.

v) When possible administer analgesics and sedatives by IV drip.

vi) Treat anticipatory pain.

vii) Provide psychological support like parental presence and music in the case of children.

viii) Anticipate cardio-respiratory complications of parenteral benzodiazepines and narcotics.

Methods of Pain Relief

1. Explanation - of the diagnosis, treatment and expected time to recovery go a long way in helping the patient understand the problem.
2. Splintage - of fractures and dislocations.
3. Dressing - of minor burns and finger-tip injuries.
4. Definitive treatment - Incision and drainage of abscesses, reduction of fractures and dislocations.
5. Analgesics - There is an enormous choice of analgesics available. The therapeutic effect varies little and the drug choice should be based on the incidence of side effects of cost of medication.

Oral analgesics are given for mild to moderate pain

a) Opioids

i) Mild pain

Codeine 30-60 mg PO q 4 hrs. Useful in fractures, burns, abscesses, headache and sprains.

Pentazocine 50-100 mg PO q 3-4 hrs. At extremes of age it may cause extrapyramidal symptoms and in addicts it may cause withdrawal.

ii) Moderate Pain

Hydrocodone: 5-10 mg PO q 4-6 hrs. As it causes dizziness, sedation, nausea and vomiting, patient should not drive or operate heavy equipment.

Oxycodone: 2.5-5 mg PO q 6 hrs. Useful in fractures, burns, I & D of abscesses, low back-ache, moderate cancer pain, dental pain and headache.

iii) Severe Pain

Pethidine: 100-150 mg PO q 3-4 hrs. Should not be used in those taking MAOI. Morphine: 15-30 mg PO q 4 hrs. The dose can be increased by 25-50% every 8-12 hrs. As with all opioids, caution must be used in patient with obstructive uropathy, Addison's disease, hypothyroidism and renal failure. Antincholinergic effects of antihistamines, tricyclic antidepressants and phenothiazines may be potentiated.

Methadone: A very long plasma half-life of 24-48 hrs. makes it unsuitable for ER use.

Buprenorphine - 0.2-0.4 mg sub lingually because of its long duration of action and low abuse potential is ideal for the treatment of painful conditions lasting over four hours such as low back pain, renal and biliary colic, newly diagnosed sickle cell disease.

b) NSAID's - Useful in mild to moderate pain

I)	Acetic acid Derivatives	Diclofenac	25 mg tid 75-100 mg/day
		Indomethacin	25 mg q 8-12 hrs 200 mg/day
		Ketorolac	10mg q 4-6 hrs 100 mg /day
II)	Fenamates	Mefenamic acid	500 mg stat followed by 250 mg q 6 hrs.
III)	Oxicams	Piroxicam	10 mg od or bd
		Tenoxicam	20 mg od
IV)	Propionic acid Derivatives	Ibuprofen	200-400 mg q 4-6 hrs
		Flurbiprofen	50 mg q 6 hrs or 200 mg od
		Ketoprofen	50 mg q 6-8 hrs. (Decrease by 50% in renal failure)
		Naproxen	500 mg stat followed by 250 mg q 6-8 hrs. (Decrease by 50% in cirrhosis)
V)	Salicylate Derivatives	ASA	325-650 mg q 4 hrs.

c) Acetaminophen 325-650 mg q 4-6 hrs. It is a centrally acting COX inhibitor with a tendency to potentiate the action of oral anti-coagulants and hence PT must be checked regularly.

d) Skeletal muscle relaxants - like carisprodol, chlorozoxazone, diazepam and orphenadrine. Care must be taken in using these in patients with urinary retention and glaucoma.

e) Miscellaneous

i) Antidepressant - Amitryptiline 10-20 mg or Fluoxetine 10 mg od prevent re-uptake of serotonin in select neuronal pathways and inhibit

transmission of painful stimuli. They are useful in migraine, fibromyalgia, post herpetic neuralgia and cancer pain.

ii) Anticonvulsants - Carbamazepine 50 mg bd, phenytoin, valproic acid, baclofen, fluphenazine have all been used to treat trigeminal neuralgia.

iii) Vasoconstrictors - Ergotamine tartarate 2 mg initially followed by 1 mg every 30-60 minutes to a maximum of 5-6 mg is used to treat vascular headaches.

Metoclopramide, nasal O_2, cocaine, Sumatriptan (a 5HT agonist) may all be given as adjuncts.

iv) Quinine sulphate 130-260 mg PO acts as an analgesic and muscle relaxant in the treatment of muscle cramps.

v) Acyclovir 800 mg 5 times/day prevents onset of post herpetic neuralgia.

II) **Parenteral analgesics** - are given for relief severe pain. The IV route is preferred because of quick and predictable action.

a) NSAID's

Useful in elderly or head injured patients in whom narcotics are contra-indicated.

i) Ketorolac 30-60 mg IM/IV upto 120 mg/day Salicylates displace it from protein binding sites.

ii) Diclofenac - 75-100 mg IM q 8 hrs.

b) Narcotics

i) Pethidine 50-100 mg IM/IV. Care should be taken in those taking MAOI.

ii) Morphine and methadone because of their long plasma half life are not ideal for use in the ER.

iii) Fentanyl 50-200 ug IV is the drug of choice for brief painful procedures in the ER such as reduction of fractures and dislocations, I & D of abscesses, burns dressing, ICD insertion. Alfentanil has a similar potency but a shorter duration of action. Sufentanil is ten times as potent as fentanyl and is considered unsafe for ER use.

C) Sedatives - They reduce anxiety and enhance the co-operation of the patient.

i) Benzodiazepines - Midazolam 5 mg IM/IV is a popular choice

ii) Haloperidol 5-10 mg max 50-75 mg IM/IV

III) Other modalities

a) Topical anaesthetics - Lignocaine gel, EMLA and amethocaine creams applied with an occlusive dressing are useful in treating post herpetic neuralgia. 140-300 mg for the trigeminal region and 250-500 mg for the thoracic region.

b) Trigger point therapy for myofascial pain

i) Stretch and spray technique - The muscle is passively stretched while a strain of vapocoolant is sprayed on it.

ii) Injection of trigger points with local anaesthetic 1-2 ml and then stretch the muscle.

c) TENS - Transcutaneous electrical stimulation of A fibers making passage of pain via C fibers difficult. Useful in low back-pain, cervical strain, tooth ache, torticollis, rib fracture. Should not be used in patients with implanted cardiac pacemakers and over the neck in patients with cardiac arrhythmias, seizures or cerebro vascular accidents.

d) Ultrasonic therapy

e) Short wave diathermy

IV) Local anaesthesia - Local anaesthetic techniques form an essential part of practice in the E.R. They have the advantage of being cheap, without affecting the conscious level and the analgesia aids

subsequent examination. Patient refusal, infection, bleeding diathesis, anti-coagulant therapy all preclude use of these techniques. Care should be exercised in patients who are elderly, have low cardiac output, impaired cardiac conduction, epilepsy, myasthenia gravis, hepatic or neurological disease. Consent and intravenous access should be taken and resuscitation equipment should always be at hand.

i) Local infiltration for cysts, lacerated wounds, abscesses

ii) Field block - for ragged wounds, abscesses and burns

iii) Nerve block
- a) Major - femoral for fracture shaft of the femur.
- b) Minor - Digital nerves
- c) Plexus blocks

V) **IVRA** for reduction of a wrist fracture

VI) **Topical anaesthesia** - E.g. foreign body in the eye

VII) **Polymer matrixes** incorporated with bupivacaine can be implanted for sustained release of local anaesthetics.

VIII) **Entonox:** A 50:50 mixture of O_2 and N_2O can be given to the patient to inhale. Analgesia begins in 30 secs and reaches its peak in 3-4 mins. It wears off 2 mins after it is discontinued. It is useful in the treatment of myocardial pain, hysterical over breathing and out of hospital to extricate limbs. The patient should be warned that he may feel dizzy or drowsy. The mask should form a good seal around the nose and mouth and patient should be instructed to take deep breaths. Obviously a pnenumothorax contra - indicates its use.

Paediatric pain mangement: When children come in pain to the ER, non pharmacological modalities like distraction imagery, relaxation techniques, music and re-assurance by parents should be tried.

The next step is to use

A) Oral analgesics - They are safe and effective when used in appropriate dosage,

i) Acetaminophen 10-15 mg/kg

ii) Codeine 0.8-1.5 mg/kg

iii) Hydrocodone 1.25-2.5 mg q 4 hrly

iv) Oxycodone>6 yrs 2.5 mg

v) NSAIDs Ketorolac 0.75 mg/kg

Ibuprofen 6-10 mg/kg

OTFC (Oral transmucosal fentanyl citrate) - may be given as a lollipop but can cause nausea and vomiting, transdermal fentanyl is also being released.

B) Parenteral analgesics

a) Morphine 0.10-0.15 mg/kg

b) Pethidine 0.6-1.0 mg/kg

c) Fentanyl 0.5-2.0 mg/kg

PCA (Patient controlled analgesia) using morphine 4 ug/kg/hr background infusion with 10-20 ug/kg boluses or piritramid, a synthetic opioid with kinetics similar to that of morphine has been used in children over 6 years.

C) Topical anaesthesia using lignocaine, EMLA or amethocaine creams

D) Local anaesthesia

i) Infiltration

ii) Field block

iii) Nerve block - E.g. Femoral or ulnar nerves, 1 ml of 1% lignocaine with or without adrenaline per year of age.

iv) IVRA - Advisable to avoid in children under 6 years. 15-30 ml is used depending on the age of the child.

E) Entonox - can be used if the child is co-operative and is about 3-4 years of age. If the child is monitored with a pulse oximeter and a cardiac monitor, it is most effective for wound repair, burns and splinting of fractures.

F) Sedation - An oral preparation of midazolam 0.2 mg/kg is a useful adjunct.

G) Anaesthesia - If needed is produced by IV or IM ketamine administration, 1-2 mg/kg and 5-8 mg/kg respectively. A single dose produces excellent analgesia, sedation and immobilisation.

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Quality Management in the Emergency Deparment

Dr. Mabel Vasnaik

Associate Prof. and Head, Department of Emergency Medicine, St. John's Medical College, Bangalore

The Joint Commission on Accreditation of Health Care Organizations (JCAHO) has adopted the principles of Continuous Quality Improvement (CQI) as a major tool used by hospitals in their performance improvement efforts. However in the 1980's and early 1990's the emphasis by the JCAHO was on Quality Assurance. This change over from Quality Assurance to CQI is because the centre piece now is on actual hospital performance.

CQI is revolutionary. It anticipates and requires a major behavioral change within hospitals and involves all individuals who directly and indirectly participate in the care delivery process.

The CQI paradigm is directly applicable to an Emergency Department ED set up.

1. CQI is internally driven not an external mandate

This comes from an increased awareness of the need of the Emergency Department to survive in the health care environment. The department recognizes its own complexity and wants to do as well as it can in providing care. This desire cannot be imposed externally. The external inspection process may intensify fear and lead to the erection of barriers to constructive problem solving activities.

CQI requires the department to take charge of its own improved performance. The Emergency Department is a process rich environment. Success requires careful sequential co-ordination of these processes.

Design and Redesign

Every process provides information by which that process can be improved. The redesign process is not a function of the management, but a function of the caregivers themselves. An active role of the providers in redesigning is essential for continuous improvement.

2. CQI recognizes that error free care cannot be guaranteed, but rather emphasizes that the quality of care can always be continuously improved.

Quality simply cannot be assured. The emergency care setting is specially vulnerable to patient expectations of error free health care. When a patient crosses the Emergency Department threshold he or she often expects perfect care in a health care setting that is one of the most difficult hospital settings to control.

Service is a Quality Standard in the Emergency Deparment.

1. For much of medicines long history physicians practiced medicine as the absolute authorities over the diagnosis and treatment of their patients. Patients were expected to comply with the authority of the physician without question. But now, the traditional role of patients and physicians is changing and patients demand to be part of the decision making process in terms of their evaluation and treatment.
2. In the quality assurance setting whenever an undesirable event occurs, the involved parties try a cover up mode, because errors are not acceptable and they need to be punished since the promise of error free care has been assumed.

   However the CQI mentality views an unfortunate event as a treasure, and brings together the involved parties for the following discussion. "How can we together improve the relevant processes of care,

so that this undesirable outcome will not occur again."

The major thrust of CQI is to improve the standard of performance rather than simply identify and punish staff when something goes wrong. Individual competence and performance is addressed by regular CME and training programmes.

3. CQI focusses on systems first and Individuals Second

Health professionals invariably want to provide good care, but sometimes fall short because of inadequate internal system support. Equipment availability, staffing,hospital policies and procedures and other factors sometimes hinder the ability to provide good care. Give all employees the proper tools to do the job right, the first time.

4. CQI is an organization wide set of activities

Multidisciplinary approach to problem solving :

The Emergency Department is a dynamic environment where patient care depends on efficient inter department interaction. Hence important issues cannot be addressed effectively solely within the department.

Individuals at all levels must cross boundaries to work with others if the improved performance objective s to be achieved. Except for the management of simple patient illnesses and injuries, effective care and good outcome from an emergency department absolutely ely on timely, effective and appropriate support from nultiple other departments.

There is a temptation to work towards creating n Emergency Department which is a hospital within a ospital or a self contained unit. However, such an pproach is an unnecessary duplication of resources nd may actually increase risk to patients because the mergency Department staff cannot provide all the skills ecessary for good patient outcome.

CQI makes good performance every ones business. All individuals from all departments contribute to the Quality care process and so they must be all involved in quality improvement activities.

Create quality improvement teams with members from all concerned departments, where by the staff can resolve issues immediately instead of waiting for the supervisor.

5. CQI requires leadership commitment

The leader should not just advocate CQI principles, but live CQI tenets by example on a daily basis.

6. CQI requires performance measurement

Precise performance measurement is required to establish baselines, identify potential opportunities for improvement and to determine whether performance has in fact improved.

7. CQI is organized around patient care, not organizational structure.

Service is a quality standard in the Emergency Department. Excellence in performance is what most Emergency Departments want for themselves and their patients. CQI offers them the opportunity to reach this lofty goal. The Emergency Department should build a customer satisfaction assessment programme.

Patients expect timely, responsible, humane, understandable and professional care.

Responsive Care : A patient should be made comfortable from the minute of arrival in the Emergency Room(ER) and the ER staff should respond to their needs.

"Deal with the patients pain and other symptoms of discomfort. If you cannot alleviate the pain immediately, explain why to the patient and make sure they understand your explanation. Give them a conservative estimate of how soon you will be able to administer medication or when they can expect to feel relief".

Quality service will differentiate E D's in the future. Departments that provide cost effective, efficient and excellent service will be the choice of patients.

Patients Complaints And Dissatisfaction

Attention to complaints by patients and their families is one of the aspects of Emergency Department Quality assurance efforts.

These complaints centre around a lack of communication, inadequate explanation of reason for delays and inadequate information about the treatment process.

Long waits are common in an EMD and a frequent source of patient dissatisfaction.

Patient satisfaction is assessed by using data and questionnaires. Examples of problems that need to be monitored in an Emergency Department are

- Changes in staff behaviour
- Avoiding delays in the ED.

1) Problem is identified
2) An assessment of the scope and details of the problem is done
3) Data is collected
4) A plan of action is recommended and implemented
5) Outcome is evaluated to see if action taken improved or reduced the problem.

Quality and Clinical Decision Making

The quality of emergency care is determined by two chief factors.

a) the quality of the decision that determines what treatment is undertaken and

b) the quality with which that treatment is executed.

If the wrong treatment is chosen, the quality of care that results will not be optimal, regardless of how skillfully treatment is executed.

Standards Development

The primary motivating force for standards development by emergency physician has been the desire to establish an acceptable level of care within the speciality. Any standards developed for emergency medicine must be complaint specific rather than diagnosis specific.

Any clinical policy however, cannot replace the treating physicians judgement.

CARE MAPS have been designed to streamline history, examination, investigations, treatment plan, timing, documentation and billing. Care maps ensure that junior residents or doctors from other specialities do not miss out on a finding.

Set goals for the Emergency Department and identify *quality improvement projects*. Evaluation of continuous improvement must be carried out.

Some Quality Indicators for the ED

1) Number of Complaints, Patient Satisfaction
2) Physician /Nurse documentation
3) Deaths in the department
4) Invasive Procedures, consultant performance
5) Cleanliness, equipment function
6) Triage, registration, billing, discharge, timeliness and accuracy.
7) Patient going against medical advice
8) Obtaining laboratory tests/ECG/radiographs

Important Tenets of CQI

1. Improve service continuously and forever
2. Institute training and retraining of all personnel
3. Institute leadership. Drive out fear, encourage communication and productivity.
4. Break down barriers between staff, encourage different departments to work together (team approach) for the good of the patient.

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Telemedicine - "The New Concept in Reaching out to an Emergency"

Brig NK Manchanda
Deputy Director General Medical Services (Army), Army Headquarters, New Delhi

Introduction

Rapid technological advancement in modern medicine enables physicians and surgeons to perform miracles in managing medical emergencies. Reports abound regarding re-implantation of severed limbs, revival of cardiac arrests and similar cases in emergency medicine. Despite all technological advancement, time still remains the most vital factor in the successful management of medical emergencies. The gap between life and death in such case is very small and can only be bridged by fast administration of medical aid. Institution of medical care or first aid at the earliest after the onset of an accident or medical catastrophe increases the success rate of revival of such cases, thereby facilitating their recovery and rehabilitation. Emergency services attempts to bridge this gap between life and death and telemedicine support can easily fill in this void by aiding in the institution of medical aid or on the arrival of paramedical staff or before reaching institutional emergency medical services.

Overview of Emergency Department

From a small "accident room" in the earlier days o the 'Casualty' or 'Emergency' equipped with ophisticated gadgets, the emergency department has ome a long way. The inexperienced residents who nanned the accident room has been replaced by pecialists in emergency medicine with facilities to urther summon highly specialized medical teams. The ngle room facility has been transformed and now as several supporting services available. The main eason for the expansion of the emergency medical services has been the large percentage of patients who are admitted through emergency services. Up to 16-30% of admission come through the emergency department.

Non-availability or non-institution of early or timely medical aid in medical emergencies remains the biggest hurdle in the success of such challenges. Delay in the institution of such aid can be the reason for sure death despite the availability, later, of super sophisticated facilities and excellent professional expertise. It is this thin gap between life and death that telemedicine can establish itself in, which will expectedly result in better success rates.

Futuristic Scenarios

1. Sepoy Dharmachand is holed up in a bunker on the border along with his section defending against a fearsome enemy attack. His morale or 'will to fight' is high as he is aware that instant medical aid is available in the form of the jacket that he was wearing. In the event of his becoming a casualty, the Warrior Personnel Monitoring System (WPMS) in his jacket will instantly establish a telemedicine link up with the nearest forward medical echelon. Monitoring of his vital parameter and expert medical advice from the specialist at the forward medical echelon will guide the nursing orderly present in the bunker to administer expert medical aid within the golden hour. This would enable early stabilisation of Sepoy Dharmchand's medical condition thereby facilitating his rear ward evacuation and saving of life and salvaging of limbs.

2. Krishna Rao is a resident of a remote and isolated village in south India. Of late he has had a few sleepless nights and some difficulty in his breathing. Soon after his lunch on the third day his discomfort worsens. He is taken to the local telemedicine centre where a technician asks Rao a few questions, as he hooks him to several probes. The instruments record Rao's vital parameters and send the data to a specialist located at a large civil hospital 300 kilometre away. An audio alert instantly draws the attention of the specialist, who finds the just received data and symptoms received on his computer unpleasant. The specialist immediately connects himself through the hospital computer to the telemedicine centre at the remote village. His face and voice appear on the telemedicine centre computer screen. The specialist then advises the technician to take an X-ray of Rao's lungs. The computer of the specialist then retrieves the X-ray image. The image is transferred to the hospital radiologist. The radiologist reads the X-ray, and inquires whether the telemedicine centre is equipped with a spirometer and pulse oxymeter. The answer is yes, of course. The technician instructs Rao to blow into the machine. He also completes the oxymeter test. The result now available to the specialist enables him to give an instant diagnosis of acute severe asthma. Bronchodilators are advised and relief is immediate. The specialist then advice Rao to be admitted to the nearest medical facility where his condition could be closely monitored. Thanks to telemedicine Rao's life is saved by telemedicine due to saving of precious time - the vital gap between life and death.

3. Cyril Contractor a corporate executive had just returned from a luncheon meeting. The time was around 3:30 in the afternoon as he entered his chamber on the thirty-fifth floor of his company's HQ located on the outskirts of the bustling metro. Suddenly Cyril's steps faltered as he felt a vice like grip across his chest. As he staggered into his chair he felt tingling numbness spread across his left arm. He loosened his tie as he found it hard to breath. Cyril realized that something was seriously wrong and summoned his secretary over the intercom. Rita, his secretary, rushed in on hearing the distressed voice of Cyril and found him sprawled over the couch, with prominent beads of sweat evident across his forehead. She immediately contacted Cyril's physician at the multi speciality corporate hospital located in downtown. The nearest specialized ambulance, one of the many strategically located all over the city, was informed over a radio to reach Cyril. The ambulance with a specialized crew was with Cyril within 15 minutes. Immediately hooked on to a lifeline and oxygen, Cyril was moved into the ambulance where specialized probes were attached to him. In the 45 minutes or more time that it would take to reach the hospital depending on the traffic, Cyril's vital parameters, ECG, blood analysis was transmitted by telemedicine to the hospital. Cyril's physician seeing the reports on his computer immediately instructed the paramedic in the ambulance to administer a shot of Morphine and Streptokinase. An hour later when the ambulance arrived at the hospital, the waiting emergency staff rushed Cyril into the ICCU. His major ischaemia had been salvaged and Cyril had been pulled out from the jaws of death - thanks to the marvel of telemedicine!

What is Telemedicine?

Telemedicine has been defined as the use of IT to provide medical information and services, through the advance synthesis of telecommunication, computer technology, robotics and medical information. It is not a speciality of medicine but a means of delivering

quality health care to anyone, located anywhere at anytime. It is achievable by the use of modern telecom means, satellite linkages, telephone and high definition television, combined with IT largely though the use of computer and Internet.

By the help of telemedicine all medical data of a patient could be transferred from a station in a remote area to a central location where expert care and opinion are available. A patient in a remote area could go to the nearest medical facility from where all his relevant documents are transferred to the major medical centre immediately by telemedicine. After reviewing these records the experts at the specialist facility could transmit back to the remote centre their advice. If necessary, video teleconferencing between the patient or his doctor and the experts at the major hospital could further facilitate the process.

Applications of Telemedicine

Telemedicine has wide scope keeping in view the large rural population with limited access to specialist medical services. Geographical isolation need no longer be an insurmountable barrier to the basic needs of timely and quality medical care. The immediate beneficial effects are the following:-

(a) Population situated in remote localities can avail of immediate expert medical advice at their place of residence and this would save them time and money towards travelling to the places where the experts or speciality hospitals are situated.

(b) Visits on indistinct ground to the experts can be avoided, thereby saving travel expense as well as time of both experts and patient.

(c) Emergency cases would get timely advice thereby the complications due to late reporting to experts would be avoided.

(d) Reduce the need for consultants and health workers to travel to visit patients located at large distance.

(e) Patients or relatives of patients can access telemedicine sites on the Web in the case of medical emergencies at home to administer appropriate first aid to stabilize the patient before transferring him to a hospital.

(f) Mass casualties at disaster or air or rail accident sites can easily be linked up with specialist centres through mobile telemedicine terminal for providing on site expert medical care.

The above can be achieved by establishing telemedicine network between PHCs, District hospitals, Zonal hospitals, Medical College hospitals and Research and Referral Centres.

Project Telemedicine of the Indian Army

In the Army the application of telemedicine assumes great significance as the more serious casualties occur closer to the border areas where adequate medical facilities and expertise are not available. Thus providing specialist opinion by telemedicine to the combat para-medic on the battlefield would lead to conserving of more trained manpower.

The 'Project Telemedicine' of the Army envisages three phases:

(a) **Phase I.** In this phase, a representative network will be created. The existing medical equipment although not directly integral, will be used. This phase will be completed in one year. The sites identified for the pilot project is as under:-

  (i) A mobile hospital for field.

  (ii) A mid zonal hospital.

  (iii) A Zonal Hospital.

  (iv) A Command Hospital

(b) Phase II. Upgradation of Phase I network with state of art medical diagnostic equipment and this phase is likely to take another one year.

(c) **Phase III**. In this the network will be further expanded to cover a large number of field medical units, designated Zonal Hospitals, 5 to 6 Command Hospitals and a hub at Army Hospital (R&R), Delhi Cantt. Connectivity to National Telemedicine Centre as well as International Telemedicine Centres could be established through this Gateway Terminal. This is likely to take 3 to 5 years.

Requirement for Telemedicine Project

The requirement for a telemedicine project are the following:-

(a) Digitisation of the medical information textual, graphical or electronic, by computerized imaging systems.

(b) Transmission of the digital information over established networks and relay over long distances by satellite transmission, if required.

(c) Interconnectivity with other networks and Internet

(d) Reception of medical information in audio and visual format to facilitate interpretation by the expert at the advisory centre.

Telemedicine technologically has two basic formats as is in vogue today:-

(a) Stored and forward

(b) IATV

Stored & Forward

This format is used for transferring digitized images from one location to another for diagnosis or consultation by capturing high-resolution colour or gray scale images and transmitting them over standard telecom lines. Scanners, digital cameras, video devices and other medical imaging systems can generate the images. Special image format prevents other programs (such as paint programs) from opening and altering the images. To ensure confidentiality data can also be encrypted when being transmitted over the network and corresponding devices at the receiving end can decipher the encrypted data.

IATV

Two ways Inter-Active Television (IATV) is used for consultations between a patient from his home or remote location and primary care provider or specialist, or for consultation amongst specialists by video teleconferencing. This format also has application in health education too.

Barriers

There is great reluctance to adopt telemedicine due to administrators not envisioning the overall impact of the technology on health care and the reluctance of medical personnel in general to be associated with IT. Further, due to non-availability of matching telecom technology often telemedicine practice cannot be adopted despite the will to do so. Moreover, upgradations of technology is an ongoing process in the field of computers and these are at times economically prohibitive. With budgetary constraints, adoption of such newer technology with high initial cost, is difficult to justify.

Contentious Issues

Accreditation & licensure

Telemedicine creates conflicts over the rules and regulations of accreditation and licensure because of the availability to medical opinion from any one over the Internet located in any country. Norms therefore have to be established that the specialist providing the opinion should be accredited and have licence to practise medicine at the place where the patient is located. By this the choice of the patient would however gets restricted and his freedom to choose his doctor over the web curtailed.

Accountability and Liability

The belief that this technology lowers liability may not be entirely true. Since telemedicine consultation involves two practitioners working together, they may be more comprehensive than traditional examination. Further physician with access to electronic medical database may find their diagnostic and treatment expertise enhanced leading to better patient outcomes. However there is evidence that as the use of technology increases so do malpractice claims, as there is disproportionate increase in patient expectation. Therefore, practitioners would be required to upgrade their new technology appropriately. Failure to correctly calibrate an instrument can increase the likelihood of inaccurate diagnosis. Deficiency or failures in equipment required to transmit an image, video clip or patient record may increase liability. In view of the physical distance between the patient and the doctor, modalities for determining the physician-patient relationship need to be established, including for non-traditional means such as an emergency care hotline or an Internet discussion group. Physicians wishing to limit involvement may have to define the limits of their participation to the patient up front. Despite the above, because even state-of-the-art technology quickly becomes outdated, it is yet unclear what obligations practitioners have to upgrade their systems periodically.

Confidentiality

Computerization can reduce some concern of concern about privacy in patient data. It increases the quantity and availability of data and enhances the ability to link the data, raising worry about new demands for information beyond those for which it was originally collected. The potential for abuse of privacy by trusted insiders is of particular concern. The problem though is not insurmountable and can be overcome by restricting the access to it by requiring authentication procedures.

Ethicality of Telediagnosis

As much help as telemedicine may be, there is no substitute for being in the room with a patient. Too much reliance on remote diagnosis or assistance can result in inaccuracies and is therefore unethical. Further, whether a diagnosis made via telemedicine will be held to the same standards of care as one made in person is debatable and will depend on available alternatives, sophistication of technology and patient's expectations.

Legal Aspects

The validity of any medical advice is a signed document by the physician. The same is as yet not possible over networks. However modalities for this need to be worked out to enable such opinion and documents to have legal sanctity.

Conclusion

Time is of essence in the management of medical emergencies. The thin line demarcating life and death is indistinct and needs aggressive and timely medial aid during the golden hour for a high success rate in the management of such cases. Telemedicine if available during this golden hour, can reach out to an emergency and provide the specialist advice and pull the victim out of the jaws of death.

Medical Negligence

Dr. Suganthi Iyer
Dept of Accident & Emergency, P.D. Hinduja National Hospital, Mumbai

Negligence - Examples

- A doctor injects an antibiotic in a child's gluteal region & the patient develops sciatic nerve damage.
- A surgeon amputates the wrong digit of a patient's hand.
- A radiologist gives X-ray burns to the patient.
- A physician is summoned & there is mismatched blood transfusion.
- An antibiotic is injected & the patient goes into anaphylactic shock.

These are some examples of negligence we read in the newspapers daily

Are the doctors only healers or merchants of health care?

Have the doctors stopped caring for their patients because there is rapid industrialisation of medicine & competition within the profession?

Have the doctors turned against each other in professional jealousy or are the doctors thinking that they could do as they please because patients are poor?

Definition

Negligence in law is defined as "if a person acts without reasonable care towards another person to whom he owes the duty of taking care & if the damage thereby caused to that person, an action in the Court of Law may result in damage being awarded for injury". The omission to do something which a reasonable man would ordinarily do or the doing of something which a reasonable and prudent man would not do is negligence.

Classification

1) Civil Negligence

- It is breach of duty to use reasonable skill & care resulting in damage.
- These are governed by the common Law of Tort.
- The patient/relatives can bring an action in a Court of Law for damages suffered by patient.
- The burden of proof is on the patient/relatives.

2) Criminal Negligence

- The degree of negligence is such that the Medical Practitioner may have caused the death of the patient.
- It is a criminal offence & governed by Criminal Law.
- It is a state which seeks redress & punishment for the wrong-doer.
- The proof has to be established beyond doubt that the accused acted with utter disregard to life & safety of the patient with deliberate recklessness.

Contributory Negligence

- The damage to the patient is caused partly by his own & partly by the doctor's negligence.
- The burden of proof rests on both the sides.
- The patient is not allowed to claim full damages.

Proof of Negligence

- To prove negligence, it must be established that the existence of duty of the defendant (doctor) to take care of the plaintiff (patient).

- Breach of the duty by the defendant (failure to attain standard of care).
- Damage suffered by the plaintiff connected with the breach of duty to take care.

Negligence is no negligence if no damage has been suffered by the patient.

An error of judgement by itself does not amount to negligence provided the doctor possesses a reasonable degree of care & diligence.

The damages may be physical, mental & financial.

The 4 D's essential in any case of negligence are:

1) Duty to exercise skill & care.
2) Dereliction of that duty.
3) Damage to the patient.
4) Direct correlation between the deficiency & damage.

1) *Duty to exercise skill & care:* The duty exists from the moment of establishing a doctor-patient relationship. This exists even in cases where a doctor approaches a patient on the roadside and the patient is unconscious.

   This is also implied in the case of pathologists & radiologists who never see the patient. The essential elements of doctor-patient relationship are willingness to examine, diagnose the case and treat the patient. This is irrespective of whether payment has been made or not.

The duties are as follows:

- Duty to be cautious.
- Duty to undertake to give treatment.
- Duty to keep knowledge update.
- Duty to diagnose properly.
- Duty to attend.
- Duty in giving injections.
- Duty in giving anesthetic.
- Duty to prevent infection.
- Duty in prescription of drugs.
- Duty to inform.
- Duty to take informed consent.

The physician owes a duty to act with utmost faith. If he cannot accomplish a cure or the treatment will be of no benefit, he must refuse to give treatment. The doctor's duty is to be very careful in diagnosing the disease & acquainting himself regarding the treatment to be given or operation to be performed. It is the duty of the physician to make true and full disclosure as to the illness / treatment.

It is also his duty to take informed consent of the patient and exercise reasonable skill, care and diligence throughout the treatment. The situations where a doctor-patient relationship is not automatically established are:

a) Examination for insurance purposes where a patient cannot claim any damage from the doctor for the loss that he may suffer due to health conditions as a result of this report wherein a situation of breach of contract is created between the doctor and the patient.

b) Medico-legal examinations like
   - Assessment of injuries in cases of assault.
   - Evaluation of disabilities for the purpose of compensation.
   - Assessing drunkenness.
   - Examination of cases of sexual offences.
   - Assessment of age for employment.

Here the doctor is not the healer & hence the question of duty of care does not arise.

2) *Dereliction of duty:* This is based on the principle of failing to do something which a reasonable practitioner would do or doing something which a reasonable practitioner would not do under similar set of circumstances. Some general concepts are as follows:

- A physician claiming to be a specialist must meet the standards of practice in that speciality.
- A physician does not guarantee cure or results. He merely exercises a degree of skill and care commonly possessed.
- A physician, if not competent to handle a case should invite consultation.
- A physician, once having undertaken care of a patient should not abandon him.
- A physician should employ the necessary and accepted method of diagnosis & treatment.
- An honest mistake by the physician is not negligence.
- A doctor is responsible for the safety of the patient/visitor in his premises. He should see that the premises are safe for the visit.

Examples of Negligence

- Failure to obtain informed consent.
- Failure to employ necessary diagnostic procedures in laboratory & imaging, specially in cases of injuries.
- Failure to inform patient about side effects of drugs.
- Failure to sterilise instruments.
- Failure of communication between two physicians handling the same case.
- Withdraw active clinical care without giving adequate notice.

3) *Damage suffered by patient:* The damages suffered by a patient are:

- Loss of earning.
- Additional expenses for treatment of complications.
- Loss of limb and reduction in life expectancy
- Damage by mental trauma.

4) Direct causation (e.g. a patient injuring his hand in an accident & alleging that the deformity developed subsequently as a result of faulty treatment). There has to be evidence that the deformity could have resulted from original injury or subsequent treatment.

- Res ipsa loquitor (fact speaks for itself).

a) Injury caused is of a nature that even a layman can recognise.

b) Procedure causing injury is under direct control of the physician.

c) Person injured is not guilty of contributory negligence.

Defences against Negligence

1) Actual denial: This is possible only when the doctor is absolutely certain about his action and has maintained accurate records.

2) Delegation of duty: In case of negligence on the part of non-medical staff, the hospital takes full responsibility.

3) Contributory negligence: This is conduct on part of the patient contributing as a legal cause to the harm he has suffered which falls below the standard to which he is required to conform for his own protection.

4) Assumption of risks: This is the doctrine of volunti non fit injuria or assumption of risk which means that a patient knows & understands the risks involved in a particular procedure.

5) Emergency: The burden of proof in such a case is on the doctor to establish that he has acted in an emergency (e.g. fracture of ribs In a cardiac massage).

6) Release of Tort Feasor: This is the defence adopted when a patient is treated for injuries as a result of an accident caused by a third person. The settlement of a suit against a third person will also relieve the doctor. If there is altogether another injury caused by the doctor, he is liable to face action.

7) Res judicata: This is the doctrine of the thing has been decided. If a doctor sues the patient for non payment of the bill where the patient has a knowledge that negligence has occurred on the part of the doctor, and does not plead this at the time of the trial then he cannot sue the doctor at a later date. Once a case has been completed between two parties, it cannot be tried again between the same parties.

8) Statutes of limitations.

9) Immunity to charitable and government institutions.

Prevention against Negligence: Prevention is better than cure.

1) Do not criticise another doctor.
2) Employ qualified staff and associate with good partners.
3) Update your knowledge.
4) Keep accurate and complete records.
5) Obtain valid consent.
6) Establish good professional relationship.
7) Take reasonable skill and care.
8) Guard against therapeutic hazards.
9) Good conduct of the doctor.
   - Doctor should have knowledge of the law and the code of ethics.
   - He should not handle a patient under the influence of alcohol/narcotics.
   - Do not practice deceit.
10) Maintain professional secrecy.
11) Relationship with legal profession.
12) Medical indemnity insurance.
13) Counter suits against the patients.

Selected Abstracts

Legal Aspects of Emergency Medicine

Dr. Suganthi Iyer
P. D. Hinduja National Hospital

I) What is meant by "Emergency"?

The word "emergency" means a sudden unexpected happening or sudden unforeseen occurrence or condition where there is a question of life and death.

II) Can refusal of medical care to injury and emergency constitute negligence?

Refer Case *Parmanand Katara Vs. Union of India* {1989 ACJ 1000 (SC)}

The Supreme Court observed:

1) There are no provisions in the Indian Penal Code, Criminal Procedure Court, Motor Vehicles Act, which prevent doctors from promptly attending seriously injured persons and accident cases before the arrival of the police.

2) The treatment of the patient would not wait for the arrival of the police for completing legal formalities. All Govt. Hospitals, Medical Institutions should be asked to provide immediate medical aid to all the cases, medico-legal or not.

3) There can be no second opinion that preservation of human life is of paramount importance. That is so on account of the fact that once life is lost, the status quo ante can not be restored as resurrection is beyond the capacity of man. Article 21 of the constitution casts the obligation on the State to preserve life. No law or state action can intervene to avoid or delay the discharge of the paramount obligation cast upon members of the medical profession.

In the case of *Paschim Banga Khet Mazdoor Samity and Ors. Vs. State of West Bengal and Anr.* {1996(4) Supreme 260 : AIR 1996 SC 2426 : 1996(4) SCC 37 : JT 1996 (6) 43 (SC)}, Hakim Shaikh, was taken to a Primary Health Centre and referred to NRS Medical College Hospital where he was refused admission as there was no vacant bed. He was then taken to other State Hospitals who also refused admission on ground of no vacant bed and no facility of Neurosurgeon and ultimately was admitted to a Private Hospital. The Supreme Court said that there was breach of the right of Hakim Shaikh guaranteed under Article 21 and the state could not avoid its responsibility for denying of constitutional right. The Supreme Court awarded Rs. 25,000/- as compensation to Hakim Shaikh.

III) Can trauma (accident and injury) patients be categorised in some way so as to determine the degree of urgency (promptness) required of the doctor to operate/provide treatment?

Yes, there are 3 categories of patients as quoted in the case of *Harbhajan Singh Vs. Dayanand Medical College and Hospital and Anr.* {1996(3) CPJ 263 : 1994(1) CPR 518 (Punjab SCDRC)}.

The 1st group includes injuries that interfere with vital physiological function and therefore immediately threaten life such as obstruction of an airway or bleeding from a gunshot wound. The primary treatment being, to establish an airway and to control the bleeding.

The 2nd group of patients are those with injuries which offer immediate threat to life and include patients

who have received gunshot wound, stab wounds, blunt trauma to the chest and abdomen but whose vital signs are stable. The majority of injured patients are in this category. Although they may require surgical procedures within 1-2 hours, there is time for additional information to be obtained.

The 3rd group of patients is those whose injuries produce occult damage. This group is composed primarily of patients who have sustained blunt trauma to the abdomen which may or may not require surgical intervention and in whom the exact nature of the injury is not apparent. These patients usually have time for extensive laboratory studies and other work-up.

TRIAGE means allocation of injured patients into certain categories, a common scheme being as follows:

1) **Critical:** Within seconds (e.g. acute laryngotracheal obstruction).
2) **Immediate:** Within minutes (e.g. tension pneumothorax or major arterial or venous hemorrhage).
3) **Urgent:** Within the "golden hour" (e.g. major burns or multiple organ injuries).
4) **Deferred:** As soon as practical (e.g. injury not life threatening or patient beyond hope of recovery or already dead).

The accurate judgement of the category has to be done by a senior and experienced clinician. In a hospital triage is used in 3 situations:

a) on arrival in the Emergency Dept.

b) in the Radiography Dept.

c) in determining priorities for operative intervention.

IV) In cases of accident, injury and emergency cases, after providing necessary first-aid, patient is referred to the higher centre, but the patient dies during transport. Will this amount to negligence on part of the referring doctor or hospital?

No. If after providing medical care upto his level of competence the doctor makes a proper referral note and refers the patient to a higher centre and any mishap occurring during transport would not be the liability of the doctor. Rather, delay in referral by the doctor could constitute negligence. Remember, not to forget to inform the police if it is a medico-legal case.

V) No consent needed for emergency operation.

Dr. T.T. Thomas Vs. Elisa, AIR 1987 Ker 52 : 1987 ACJ 192 (Ker HC).

Doctor not performing an emergency operation for want of patient's consent and consequent death of the patient. Patient was admitted to the hospital and the operation was not performed on the very day and on the subsequent day and the patient died owing to bursting of the appendix. Patient's relatives' case was that, had the operation been performed immediately, the patient's life would have been saved. Defendant Surgeon's plea of refusal by patient to give his consent to the operation, which was the reason for non-performance of surgery on the very first day, was not accepted by the Court and compensation of Rs. 37,000/- was awarded. The burden of proof that consent was sought and it was refused was upon the one who takes up such a plea.

A doctor can lawfully operate on or give other treatment provided it is in the best interest of the patient if it is done in order either to save his life or prevent deterioration of health. In cases of emergency or unconsciousness, all considerations regarding consent will be set aside and doctors will do whatever is necessary to save the life of the patient.

A patient's interest consists of his right to self-determination. Society's interest is in upholding the

concept that all human life is sacred and should be preserved, as far as possible. In a situation where these two interests conflict, the right of the individual is paramount. But where there is doubt, the doubt falls to be resolved in favour of preservation of life.

VI) Cases to be intimated to the Police.

1) Road Traffic Accidents.
2) Poisoning
3) Trauma (e.g. machinery)
4) Burns
5) Unconsciousness of unknown origin
6) Physical abuse
7) Attempted cases of suicide, homicide
8) Child abuse
9) Prisoner
10) Mentally unfit person
11) Person in custody of police
12) Post-operative cases where surgery has been done elsewhere and case transferred here.
13) Mismatched blood transfusion

VII) Repeal of Coroner's Act, 1871 by Ordinance No. XVII/1999, dt. 26-7-99.

The new guidelines are as follows:

1) A person dying in a hospital even within 24 hours of admission if there is no suspicion of foul play and the Medical Officer is sure of the diagnosis, the Death Certificate can be issued.

2) Even if a death occurs at home and the Medical Officer is capable of certifying the cause of death as to the cause of death being a natural one and no foul play suspected, no medico-legal formalities are required.

Govt. of Maharashtra has published a set of instructions to be adopted and all cases of accidental, unnatural or suspicious deaths are envisaged in Section 174(i) or 174(iii) of Criminal Procedure Code and have to be reported to the nearest Police Station. The divisional ACPs of the region have been empowered and designated as Executive Magistrates in place of the former Coroners/Additional Coroners and Deputy Coroners. They have to be intimated and are in charge of subject matters related to any cases about the death as required under Section 174 of the Criminal Procedure Code.

The information regarding an accidental, unnatural or suspected death where the Medical Officer attending on the deceased is not in a position to ascertain the cause of death should be registered to the nearest Police Station. The Station Officer at the Police Station in turn is supposed to reduce such information received in Form I and file in the station diary. On receiving such information, the local Police Station deputes the investigating officer to take charge of the body. All the Officers of and above the rank of Inspector are empowered to perform the duties of holding inquest in cases of death under question. On a detailed inquiry and findings in the cases of unnatural and suspected deaths should be sent for post-mortem and examination by the Police. The Govt. of Maharashtra has started 8 post-mortem centres all over the city of Mumbai at J.J. Hospital, Nair Hospital, KEM Hospital, Lokmanya Tilak Hospital, Rajawadi Hospital, Cooper Hospital, St. George Hospital and Bhagwati Hospital. The body should be accompanied by a brief history of the chain of events leading to the death and the line of treatment offered by the hospital or the attending Medical Officer. All the Medical Officers and Post-mortem Centres are under the technical and administrative charge of the Police Surgeon of Mumbai. In cases of difficulties regarding the post-mortem work, non receipt of provisional cause of death or post-mortem notes, non-availability of Medical Officers to perform post-mortem or refusal to perform post-mortem at odd hours, etc., the Police Surgeon should be immediately contacted for immediate necessary

assistance. After the post-mortem examination is over, the body should be handed over to the claimants by the Police along with the Death Certificate in the format of FORM-IV bearing the signature of the Inquiry Officer with time, date and stamp of the concerned Police Station which implies that there is no objection to the disposal of the body. The claimants have to sign the receipt of the body in FORM-V. Some of the special cases like death of a newly married woman or deaths of women within 7 years of marriage, especially deaths related to burns bear a special significance and the Medical Officers should be very careful before certifying the cause of death. In case of suspected foul play, the police should be informed.

VIII) Dead bodies which have to be referred to Police before disposal?

1) All medico-legal cases

2) A dead body within limits of Greater Mumbai and there is a reasonable cause to suspect that such a person died a sudden death of which cause is unknown or is not adequately or satisfactorily certified.

3) If there is reasonable suspicion that:

   a) Death is caused by suicide, homicide, infanticide (i.e. death caused by violent means).

   b) Death was caused by accident, poison, machinery.

   c) Death was caused by an occurrence arising out of use of vehicle in the street, public road or private place (includes death caused by perambulator, bath chair, train, bicycle, etc.).

   d) If cause of death is certified or given as "post-operative shock" or "post-operative bleeding" or "hemorrhage".

   e) If death occurred in circumstances, the continuance or possible recurrence is prejudicial to the health or safety of the public or any section of the public (this includes deaths due to rare infectious diseases or on operation table).

4) If death occurred in prison in which deceased was prisoner or it occurred when deceased was in custody of police, or that death occurred:

   a) In a leprosy asylum

   b) In an asylum or mental hospital

   c) In a Borstal School

   d) In a Receiving Centre or Certified Institution provided and maintained under the Bombay Beggar's Act.

   e) In any Certified School, Remand Home or an institution or approved place, established, maintained, declared or recognised as the case may be, under the Bombay Children's Act.

Unless in these and similar cases, the Police authorities are referred to for disposal of the dead body, it is an offence to dispose of the dead body.

References:

1) Medical Negligence and Compensation, 2nd Edition by Dr. Jagdish Singh, Vishwabhushan (Bharat Law Publication)

2) Law of Medical Negligence and Compensation by R.K. Bag (Eastern Law House)

3) The Maharashtra Ordinance, 1999 (repeal of Coroner's Act)

4) National Commission and Supreme Court on Consumer cases.

5) Bombay Municipal Corporation Act.

Predictors of Mortality in Patients Admitted Through Emergency Department

Dr. Shashiraj E.
Dept of Emergency Medicine, St. John's Medical College, Bangalore

Introduction

Various scoring systems are available to predict mortality in intensive care units. Most of these are developed in western population and may not be directly applicable to Indian population. Some of these systems are tested and validated in Indian intensive care units. One such system is Logistic Organ Dysfunction (LOD) System. In this study LOD scoring was used to predict mortality in patients admitted through Emergency Department after initial stabilization.

Methods

One hundred consecutive patients who spent 24 hours or more in the Intensive Care Unit (ICU) and Intensive Therapy Unit (ITU) attached to department of Emergency Medicine before being shifted out to other departments were studied prospectively. LOD was applied to all these patients on the day of admission and the discharge outcome was analysed.

Results

The observed mortality rate was 38%. It includes discharges against medical advice as many of these patients were critically ill and were taken home by the relatives due to various reasons namely financial constraints, bad prognosis etc.

Table 1: Patient Characteristics

No. studied:		100
Age (in Years):		15-84 (Mean: 43)
Sex Male : Female		63 : 37
Discharge Outcome:	Alive	62
	Dead	38*

(*includes 15 patients who were discharged against medical advice)

Table II shows survival and mortality rates at different levels of LOD scores

Table II: Discharge outcome at various LOD scores

LOD	n	Alive %	Dead %
0-3	61	52 (85.2)	09 (14.8)
4-7	14	07 (50.0)	07 (50.0)
8-11	12	03 (25.0)	09 (75.0)
12-15	08	0 (00.0)	08 (100)
>16	05	00 (00.0)	05 (100)

Standardized mortality rates (SMR) for the observed mortality rates were calculated using the hospital morality predicted by the regression equations proposed by the Le Gall et al. and Sampath et al. separately. These are shown in Tables III & IV.

Table III. Observed mortality Vs mortality predicted by Le Gall et al.

LOD	n	Observed Mortality	Expected Mortality (Le Gall et al.)	SMR
0-3	61	09	3	2.7
4-7	14	07	4	1.81
8-11	12	09	7	1.26
12-15	08	08	7	1.11
>15	05	05	5	1.02
Total	100	38	26	1.44

Table IV. Observed mortality Vs mortality predicted by Sampath et al.

LOD	n	Observed Mortality	Expected Mortality (Sampath et al.)	SMR
0-3	61	09	8	1.06
4-7	14	07	6	1.11
8-11	12	09	9	1.03
12-15	08	08	7	1.08
>15	05	05	5	1.02
Total	100	38	35	1.06

It is evident from the results shown in Table IV that observed mortality rates are similar to the hospital mortality predicted by regression equations proposed by Sampath et al. (P=0.068). But the mortality predicted by the regression equations of Le Gall et al. is low (26%) compared to the present study (38%), but the difference observed is not statistically significant (P=0.76) (Table III).

Discussion

Both the LOD models (Le Gall et al. and Sampath et al.) had good correlation and are useful in predicting outcome of patients admitted through the Emergency Department. These help in assessing prognosis of patients admitted to Observation Units or Short Stay Wards attached to Emergency Departments.

References

1. Le Gall J.R., Klar J., Lemeshow S., Saulnier F., Alberti C., et al. The logistic organ dysfunction system: A new way to assess organ dysfunction in the intensive care unit. ICU scoring group. *JAMA* 1996; 276:802-10.

2. Sampath S., Fay M.P., Pais P. Use of the logistic organ dysfunction system to study mortality in an Indian intensive care unit. *Natl Med J India*. 1999; 12:258-61.

Patterns and Presentations of Emergency Department Visits a Community Hospital Experience

Dr. Deepa Doraisamy, Dr. M.Pai, Dr. G. Bhuvaneswari, Dr. Arjun Rajagopalan, Dr. Kumar Alagappan
Department of Emergency Medicine
Sundaram Medical Foundation-Chennai & Long Island Jewish Hospital - New Yourk

Introduction

Emergency Medicine being a relatively new speciality in our country - this study was done with the objective of finding out the descriptive epidemiology of ED visits at our hospital which has full faculty, well organised 7 bedded Emergency Department. Several parameters including the average no. of ED visits, case mix, type of patient population, common diagnostic groups, time frames, investigations done, outcomes were analysed.

Methods

Setting: Urban, secondary care hospital catering predominantly to a middle / upper income population in Chennai city, Design: Retrospective, descriptive review of charts.

Sample: All ED visits during 4 months in 1998, randomly selected from each quarter (total of 4089 cases)

Data was abstracted from patient chart and analysed using epi-info statistical software.

Results

Total no. of visits:4089. 57.6% were females. Paediatric population accounted for 17% of the total visits. 94% of the patients were seen within one hour of their arrival in the emergency. Trauma contituted 22% of the total no. of cases, motor vhicle accidents being the commonest cause of trauma and 54% of them presented within the golden hour. Fever, trauma and abdominal pain were the 3 most common complaints. Fever for evaluation, minor lacerations and fractures were the most common ER diagnoses. CBC and eloctrolytes were most common labs ordered. 50% of the cases were discharged directly from the ER. 14% were admitted in the wards and 3% in the intensive care.

Conclusion

This study can be useful to generate baseline information for plannin uiform standardised ER care in our country

Pre-Hospital Patient Information System at a Premier Tertiary care Hospital A Record Based Retrospective Study

Dr. Sidhartha Satpathy
Department of Hospital Administration
All India Institute of Medical Sciences, New Delhi

Introduction

Developing Countries like India which have achieved some degree of industrialsiation are gradually becoming interested in "Emergency Medicine" as a full fledged speciality. Worldwide, including in India, injuries are recognised as the leading cause of premature deaths, causing immense economic loss. Historically, all over the world emergency medical service systems have originated from sytematic and methodical collection of prehospital patient data. Unfortunately, a number of local studies carried out in various hospitals in our country indicate that either no pre-hospital data is available, or wherever available, it is sketchy and no-uniform.

Methods

Pre-hospital data was collected from the records of one hundred medico legal cases attending the Accident and Emergency services at AIIMS in the month of January '2000. The same was analysed according to modified uniform EMS data elements developed in USA in 1995.

Results

It was found that two thirds of the patients were adult males whose demographic parameters had been recorded properly. Legal registers in about one third of the cases. It was found that in about 50% of cases the pre-hospital data elements were mentioned; whereas in the remaining 50% these were not recorded by the doctors posted in the Casualty.

Conclusion

There is an imperative need not only to develop uniform data elements in our country, but also to create awareness amongst EMC providers in hospitals regarding the same, in order to develop emergency medical services in a systematic and scientific manner. Subsequently, it will also be useful for system evaluation, medico legal cases and quality improvement in EMS.

Cost Evaluation of Medical Services in a Large Superspeciality Teaching Hospital in Delhi*

Shakti Gupta,# Satish Kumar,# R.K. Sarma,# P.P. Kotwal**
*Departments of Hospital Administration# and Orthopaedics,***
All India Institute of Medical Sciences, New Delhi

Introduction

Emergency Medical Services are an integral and vital component of a health care system. Rapid industrialization and increase in vehicular traffic are the major contributors to emergencies, of which the common man is often the victim. The emergency medical services encompasses the decision making and action necessary to prevent death or any further disability for patients in health crisis as well as in health promotion and injury prevention efforts. The patient population is unrestricted and presents with a full spectrum of episodic, undifferentiated, physical and behavioural conditions.

To be effective in today's dynamic health care area, health professional must constantly assess the reasons for their existence in the organization and they must plan accordingly. The political, social, economic, regulatory and competitive factors that surround the health care industry are manifested in the emergency services. The nature of cases which report to the emergency are critical and the staff has to deal with life and death episodes that demand crucial decision making. Whenever some emergency lands in a hospital, it is not only the patient's life or limb that is at stake but also at stake is the reputation and image of the hospital and treating physician. If proper attention is not paid, it can make or mar the image of the hospital.

Emergency medical services demand two important features:

- It must be efficient at all times and must fulfill the purpose for which it is meant;
- It must be cost-effective and cost beneficial.

The primary role of emergency services is to provide prompt and appropriate attention, constant supervision and adequate diagnostic facilities. The aim is to prevent death, disability and suffering of those who are injured or taken seriously ill.

Rationale of the Study

Emergency services being provided free in government hospitals, have no separate budgetary allocations. The cost is met by diverting the resources from the total hospital budget. In order to project budgetary requirement for running a smooth and viable emergency services, cost analysis of emergency services is mandatory. As no such exclusive study has been conducted at AIIMS hospital emergency services, there is absolute need for such a study. With these considerations this study was planned to analyse the cost of emergency medicare services and also to compare the expenditure incurred on selected categories of patients to help in budgetary projections and for levying of charges for emergency services if ever required in future apart from suggested measures for efficient utilization of financial resources in this area and identification of wasteful expenditure if any.

Area of Study

It is quite difficult to find out the cost incurred on each and every patient who visits the Casualty as some of them stay there for a very short period, follow up is much difficult and in others, the cost incurred may be quite negligible. It was proposed to conduct the study in the following areas so that a realistic expenditure incurred in these patients is calculated.

** The study has been conducted by Dr. Satish Kumar, MHA Resident, Dept. of Hospital Administration, AIIMS, New Delhi in the year 1998-99*

This study is restricted exclusively to those patients who:

- Receive treatment in the examination cubicles, observation ward or treatment room of the Casualty
- Undergo investigation in the Casualty only
- Operated upon in the Casualty OT only

It does not include those patients who receive indoor care provided to such patients admitted through Emergency.

Aim

To calculate the expenditure incurred on emergency services with special reference to selected categories of patients at AIIMS so as to have a separate budgetary projection.

Objectives

- To make an assessment of total workload in the Casualty and determine the disease profile of the patients
- To calculate the total cost incurred in the management of patients in the Casualty at AIIMS
- To appropriate the cost involved in selected patients' categories treated at AIIMS
- To suggest measures for maximizing the resource utilization and improvement of services

Methodology

Literature on structure and functioning of emergency services was reviewed. Methods and principles of costing applicable in health care and hospital setup, currently being adopted all over and especially in the developed countries were referred to in detail.

An observational study of one week duration was carried out in the casualty OPD to gain an insight into the physical facilities, manpower, material resources, equipment, policies and procedures being followed in the casualty OPD.

In order to workout the disease profile of patients attending the AIIMS casualty, a prospective study of the patient visits in the casualty was carried out during the month of March 1999 when the seasonal bias is the least. Thirty six shifts workload randomly selected covering all the three shift in a day, were analysed.

Total cost incurred in running of the emergency services at AIIMS was worked out by taking into consideration all the inputs required for delivery of emergency services like wages and salaries of the providers of Medical Care, Medical, Surgical, Linen, Stationery, Crystalloid, General Stores Supplies, Equipment Cost, overheads like Building Depreciation Cost, Maintenance Cost, Rental Value or Opportunity Cost, Cost of Electricity Consumption, Water, Equipment Depreciation, Comprehensive Equipment Maintenance Cost.

Outcome

The total workload in AIIMS Casualty during the year 1998-99 was observed to be 1,19,806 and is increasing at an average rate of 2.3% annum. This workload does not commensurate with the available space. As per norms, the minimum space requirement for this patient volume is 3282 sq.m. whereas only 2688 sq.m. is available.

It has been observed that Institute is spending Rs. 4.24 crores for providing emergency services out of which drugs and supplies account for Rs. 72.07 lacs and the average per patient cost out of the total expenditure is Rs. 354.18.

The expenditure incurred on the Casualty services constitutes 1.89% of the Institute budget and 31.50% of the main hospital budget.

Survey of Emergency Departments in Chennai

Dr. Deepa Doraisamy, Dr. Theresa Van det Flught, Dr. M.Pai, Dr. Arjun Rajagopalan,
Department of Emergency Medicine, Sundaram Medical Foundation,
Dr. Rangarajan Memorial Hospital, Chennai

Introduction

In Jan 2000, a survey of the emergency departments of 18 hospitals in Chennai was performed. A standards set of 157 questions was asked to the administrative officer and the CMO. Question concerned the staff of the ED, resources, medical equipments, materials etc., and the data was collected in MS excel and analysed in Statware.

Methods

A prospective study including 18 hospitals that were grouped into small, medium and large ones. 13 were private for profit hospitals, 2-corporate hospitals, 2 govt.hospitals, 2 non profit hospitals.

Results

23% of the total admissions came from the ER. Paediatric case accounted for 7 to 18% of the total. Trauma constituted 20%. The ER was staffed with 1 to 2 physicians per shift. Most of them were fresh medical graduates. 1 in 18 hospitals had consultants in Emergency medicine. No consultant level physician was trained in Emergency Medicine. The primary ER consultant was a physician followed by a surgeon. Only 3 out of 18 hospitals had personnel trained in ACLS, ATLS and PALS. 7 hospitals had no defribrillators. 3 hospitals had no ECG machines in the ER. Trauma was the most common diagnoses but only 2 hospitals had a rigid trauma board. 8 out of 18 hospitals had personnel trained in ACLS, ATLS and PALS. 7 hospitals had no defribrillators. 3 hospitals had no ECG machines in the ER. Trauma was the most common diagnoses but only 2 hospitals had a rigid trauma board. 8 out of the 18 hospitals had cervical collars. Only 13 hospitals had all the life saving drugs.

Conclusion

A high level of descrepancy was noted between the equipments reported and the equipments present in the ER. No standardisation of ER equipments or drugs were present. Some very small hospitals had very well equipped ER and some large hospitals were not so well equipped. To provide uniformity of ER care, emergency room treatment and training of physicians should be standardised in India and monitered so that it adheres to the standards of the west.

Aggressive Atropinsation and continuous paralidoxime (2-pam) Infusion in patients with severe organophosphate poisoning: Experience of a North West Indian Hospital

Surjit Singh, Dhruv Chaudhary, D Behera, Dheeraj Gupta, Surinder Kumar Jindal
Dept. of Internal Medicine and Pulmonary and Critical care Medicine, Postgraduate Institute of Medical Education and Research, Chandigarh

Objective

The aim of the study was to find whether continuous pralidoxime infusion along with aggressive atropinsation improve the outcome in patients with severe organophosphate poisoning who require assisted ventilation.

Methods

Sixteen patients admitted to our ICU with severe organophosphate poisoning and requiring assisted ventilation were included in the study. The compounds involved were phorate (6), dichlorvos (4), oxydimetion methyl (1), monocrotophos (1), methylparaben (1) and in 3 it was unknown. They were given intermittent i.v. bolus atropine 5 mg at onset and then 2.5 mg every 5-10 min. till atropinsation was achieved and then maintained either by intermittent bolus doses or by continuous infusion if the required dose was large. They were also given continuous i.v. infusion of paralidoxime in dose of 7.5 mg/kg body weight/hr (maximum 500mg/hr) after a bolus dose of 2 gm at onset.

Results

The mean (+S.D) dose of atropine was 735.02+742.98 mg (range 85-3000 mg) with maximum dose on day 1. The mean (+ S.D.) duration of pralidoxime infusion was 96.4 + 49.4 hr. (range 10-216 hr.) The mean (The mean (+S.D) duration of mechanical ventilation was 131.5 hr + 95.65 (range 4-336 hr.) Fourteen patients could be successfully, extrubated and two died of bronchopneumonia and sepsis (mortality 12.5%)

Conclusions

Continuous pralidoxime infusion along with aggressive atropinsation improved the outcome in severely intoxicated patients with organophosphate compounds who required assisted ventilation in this case series.

Emergency Help in Mumbai (EHIM) Study and Organising the Time to Time (TTT) Trial

Galwankar S, Vardhachary K. S.
Department of Internal Medicine, P. D. Hinduja National Hospital & Medical Research Centre, Mumbai

Introduction

Emergency Medicine has fast emerged into a specialized branch of medicine which involves specialized trained personnel, sophisticated equipment and efficient backup systems, all working in an extremely well organised manner. Mumbai is one of the fastest and the most busiest metropolis of India where the health care systems serve a large variety of people from various castes with varied economic backgrounds in wide variety of conditions.

Health care systems in Mumbai mainly involve efficient hospital based care. A study was hence done to evaluate the efficacy as well as the efficiency of Pre-hospital and Hospital Based Emergency Care in Mumbai and a trial was planned to apply the same.

Methods

Overall study of the social, personal, economic, legal aspects as well as the available health care system were evaluated with respect to giving emergency services to the public. A trial was then planned to apply both Pre-hospital as well as Hospital Based Emergency Care to the patients in acute stage.

Results

There was Sufficient as well as Efficient Hospital Based Care available but there was Disorganized Pre-hospital Care which was due to many factors like lack of co-ordination, awareness etc. The trial if successfully applied could provide a breakthrough in providing total emergency care right from the place where it is needed which would at least make a good beginning to Emergency Medicine in the metropolis.

Conclusion

There is a need of public awareness as well as good organization and support to apply emergency medicine protocols at the right time to the places where they are needed so that the needy can be brought to the hospital in a stabilized state to avail good hospital care after receiving good pre-hospital emergency care.

Primary Angioplasty in Myocardial Infarction (PAMI) Programme and Some Recent Success

Galwankar S, Sharma GL, Joshi M, Dalal JJ, Rajani R, Kumar N,
Department of Cardiology & Emergencies, P. D. Hinduja National Hospital & Medical Research Centre, Mumbai

Introduction

In recent years the prognosis for patients with acute myocardial infarction which is a life threatening cardiac emergency has been improved by timely restoration of blood flow in the infarct related coronary artery. PAMI has now replaced the conventional thrombolysis therapy. According to recent studies compared with thrombolysis therapy PAMI is associated with better clinical outcomes in the future.

Methods

We proposed, introduced and organised a PAMI programme right from the place where the patient had a myocardial infarction to our hospital Cath Lab which included both pre-hospital and hospital based complete emergency care. This programme has its own limitations which were also studied.

Results

Proper counselling, patient understanding, rightly timed, organised and administered pre-hospital care with efficient hospital based back-up systems can lead to saving a patient of myocardial damage and cardiac disability due to myocardial infarction. This is shown by our recent success of the programme.

Conclusion

PAMI programme is a very useful entity which has to be implemented when needed as an emergency protocol and rescue for a patient in myocardial infarction. This when done in an organised and coordinated manner is a life saving measure.

Medicolegal Cases in Emergency Medicine Department: A Retrospective Analysis

Dr. Shashiraj E, Dr. Vivian D'Souza,
Dept of Emergency Medicine,
St. John's Medical College Hospital, Bangalore

Introduction

Injuries and poisoning are one of the major causes of mortality, morbidity and disability. They have been recognized worldwide as serious public health problems.

The epidemiology of injuries and poisoning need to be documented and specific programmes for their surveilance and prevention are very essential. This study is aimed at examining the spectrum and causes of medicolegal cases which include trauma and poisoning, their relationships with demographic characteristics as well as hospital utilization.

Material and Methods

In order to study the spectrum and outcome of medicolegal cases a retrospective analysis of 1000 such cases, that had presented to the Dept of Emergency Medicine, St. John's Medical College, Bangalore, was conducted. The cases had presented to the Dept between the months of October 1998 and February 1999.

Results

During the study period, 1000 cases were analysed of which 78.6% of the patients were male. The most Vulnerable age group for both sexes was between 20 and 29 years (39.9%).

Types of Medicolegal Cases

Road Traffic Accidents accounted for the majority of the cases 48.1%. The other common cases were acute poisoning 15.6%, Industrial Accidents (13.4%), Assaults 8.4% and Burns 5.1%

Outcome of the patients

25.6% of the patients were treated and sent home. Another 19.4% of the patients were referred to other hospitals after initial treatment and stabilization.

The remaining 55% of the patients were admitted to various departments such as Intensive Care Units (11.5%), Orthopedics (12.9%), Neurosurgery (11.6%), & Plastic Surgery (8.9%)

Head Injury

Head Injury was observed in 189 cases. The commonest cause of head injury was RTA, (80 patients) Other Causes include: Fall from a height (25 cases), Industrial Accidents (6 patients) & Assault

Poisoning

Acute poisoning accounted for 15.6% of the medicolegal cases

The poisons consumed were: Organophosphorus (OP) compounds (74), Benzodiazepines (15), Anticonvulsant drugs (08) & Antipsychotic drugs (05) & Other Poisons (54) which include rat poison phenyl alcohol etc.

In the cases of OP poisoning, males accounted for 63.5% of the total number. In the other poison groups the sex incidence was almost identical. The most vulnerable group was that of young adults aged between 20 and 29 years. The majority of these cases of poisoning were suicidal (70%). 28% of the cases were accidental. 02 % were either homicidal or of unknown intent.

Industrial Accidents

Industrial accidents accounted for 13.4% of the patients. The majority of the patients were male (92.5%), aged between 20 and 29 (64.7%). The most common Industrial Accident noticed was crush injury of the limbs (42%)

Mortality

The overall mortality of the patients was 4.1%, i.e. 41 patients. Traffic related accidents accounted for 18 Deaths. The other causes include: Poisoning (7), Burns (6), Fall from a height (4), and 2 cases of death related to injures sustained while at work.

Conclusion

Our data indicates that trauma and poisoning are the major causes of mortality, morbidity and disability. The most vulnerable groups were males aged between the ages of 20 and 29 years. The leading cause of death among the Medico Legal Cases was RTAs, accounting for 48.1% of the Deaths. In fact RTAs together with Poisoning and Industrial Accidents account for more than thre-fourths of the total number medicolegal cases.

Active (e.g. Traffic Regulations, Education of drivers & riders) and Passive (e.g. use of safety helmets, and seat belts) measures may reduce both incidence and severity of injuries.

Poisoning is an alarming emergency these days. The attention of all concerned shoud be drawn to this huge, untimely and tragic loss of lives. The suggested preventive measures include stringent restriction of free supply of organophosphorus compounds, Psychiatric counseling, education of personnel involved in health care at grassroots level and establishment of 24 hour Drug and Poison information centers

Tuberculosis in the Emergency Room: SVIMS Experience

Mohan A, Sriharibabu G, Abha Chandra,* Dilip D,* Vengamma B,** C.V. Harinarayan,*** M.H. Rao****

Departments of Emergency Medicine, Cardiothoracic and Vascular Surgery, Neurology,** Endocrinology,*** Anesthesiology****

Sri Venkateswara Institute of Medical Sciences, Tirupati 517 507, Andhra Pradesh

Introduction

Tuberculosis is a major public health problem in India. The deadly synergy between human *Mycobacterium tuberculosis* and the human immunodeficiency virus [HIV] and the problem of multidrug-resistant tuberculosis [MDR-TB] have rendered health care workers vulnerable to tuberculosis. In a country like India, where the disease is highly endemic, tuberculosis continues to be a diagnostic dilemma and is often encountered in the Emergency Room.

Material and Methods

We retrospectively reviewed the clinical presentation of tuberculosis at the Emergency Service at the Sri Venkateswara Institute of Medical Sciences Hospital, Tirupati between January and December 1999.

Results

Ninety two patients with various forms of tuberculosis constituted 2.7% of all Emergency patients [n = 3349] seen during this period. One of them [1%] was HIV-positive. Their mean age was 33.7 years [SD 18.3; range 1-74 years]. They included patients with tuberculous meningitis [n=32, 35%]; tuberculous empyema [n=19, 21%]; hemoptysis [n=8, 9%]; tuberculous pleural effusion [n=7, 8%]; pneumothorax [n=7, 8%]; abdominal tuberculosis [n=6, 7%]; pyopneumothorax [n=4, 4%]; pneumonia [n=3; 3%], pulmonary tuberculosis and respiratory failure [n=2, 2%]; miliary tuberculosis [n=2,2%]; hydropneumothorax [n=1; 1%] and pericardial effusion [n=1, 1%]. Twenty of these 92 patients [22%] were "open cases" and were sputum smear-positive. Non-insulin dependant diabetes mellitus [11%] was the commonest predisposing factor. Hemoptysis was due to aspergilloma in tuberculous cavity in two patients. Atypical presentations included absence of fever [n=11, 12%]; hypoglycemia being the presenting symptom [n=2, 2%]; respiratory failure requiring assisted ventilation [n=2; 2%].

Conclusion

Atypical presentation of tuberculosis can result in diagnostic delay and therefore a delay in the institution of specific antituberculosis treatment. Health care workers in Emergency Room should follow precautions to protect themselves from tuberculosis.

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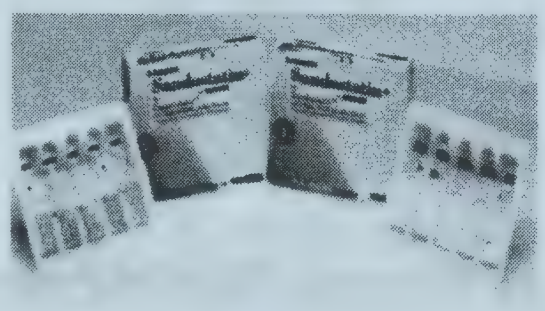

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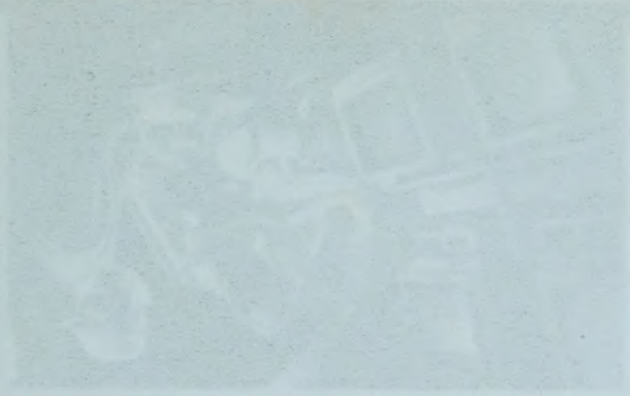

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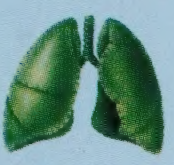

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